inflated.

The treatment of pneumothorax ex vacuo should be
directed to relieve the endobronchial obstruction. This may
consist of aggressive suctioning to remove secretions or
mucous plugs, fiberoptic bronchoscopy to extract aspirated
foreign bodies or inspissated plugs, or repositioning of a
malpositioned endotracheal tube. Chest tube placement
may not result in reexpansion of the collapsed lobe in the face
of endobronchial obstruction and is not indicated. The
diagnosis is confirmed when the intrapleural gas spontaneously
resolves after the endobronchial obstruction is relieved and
the lobe reexpands (Fig 1-3).

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Kyphosis Secondary to
Tuberculosis Osteomyelitis as a
Cause of Ventilatory Failure*

Clinical Features, Mechanisms, and
Management

Ian E. Smith, MA; Clare M. Laroche; Sarah A. Jamieson; and
John M. Shneerson, MD

Study objectives: To investigate the relationship of
thoracic kyphosis following tuberculosis to the develop-
ment of ventilatory failure and to assess the efficacy
of nocturnal noninvasive ventilatory support.

Design: Retrospective consecutive case series with
crossover from a phase without noninvasive ventila-
tory support to a phase with this treatment.

Setting: The Respiratory Support and Sleep Centre,
Papworth Hospital, Cambridge, England.

Patients: Seven patients with thoracic kyphosis follow-
ing tuberculous osteomyelitis which had been contracted
by the age of 4 years were studied. Their mean age was
53 (SD 7.1) years and the mean angle of kyphosis was
113.60. All patients were in ventilatory failure.

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Manuscript received November 6, 1995; revision accepted May 1,
1996.

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Centre, Papworth Hospital, Papworth Everard, Cambridge, En-
gland CB3 8RE.

Interventions: The patients were treated with nocturnal
noninvasive ventilation with either an individually con-
structed cuirass shell and a negative pressure pump or
nasal intermittent positive pressure ventilation using a
volume preset ventilator.

Measurement and results: Each patient underwent an
initial clinical assessment along with radiologic studies of
the spine, pulmonary function tests, daytime arterial
blood gas tensions, and overnight recordings of arterial
saturation, and transcutaneous carbon dioxide tension.
They were reassessed in detail at a mean of 5 years after
starting ventilatory support. Symptoms, vital capacity,
daytime carbon dioxide tension, and overnight oximetry
had all improved following treatment. Temporary with-
drawal of ventilatory support led to severe sleep frag-
mentation in four patients and the appearance of central
apneas and hypopneas in the other three. Six of the 7
patients were alive at a mean of 5.7 years after starting
nocturnal ventilation.

Conclusion: These results show that ventilatory failure
develop, after an interval of many years, in patients
with a severe thoracic kyphosis due to tuberculosis in
childhood. Noninvasive nocturnal ventilatory support
can control the symptoms of ventilatory failure, improve
the physiologic abnormalities, and is associated with
prolonged survival. (CHEST 1996; 110:1105-1110)

Key words: kyphosis; mechanical ventilators; respiratory insuffi-
cency

Abbreviations: DI=desaturation index (number of dips in SaO2
>4% per hour); Pemax=maximal expiratory mouth pressure;
Pirmax=maximal inspiratory mouth pressure; PtcCO2=trans-
cutaneous partial pressure of CO2; REM=rapid eye movement;
SaO2=arterial oxyhemoglobin saturation; TLC=total lung capacity;
TSaO2 <90%=percentage of time overnight when SaO2 <90%.

Over a period of 10 years, 7 patients have been referred
to the Respiratory Support and Sleep Centre, Pap-
worth Hospital, Cambridge, England, with ventilatory fail-
ure associated with kyphosis and no other respiratory disor-
der. No previous report has analyzed this association or the
outcomes of treatment. We therefore examined the clinical
and physiologic factors which might have caused ventilatory
failure in this group of subjects and present the results of
treatment with long-term nocturnal-assisted ventilation in
the home.

MATERIALS AND METHODS

Subjects
The records of all patients referred to the Respiratory Support
and Sleep Centre who had kyphosis and subsequently received as-
sisted ventilation were examined. Patients with an associated scoli-
osis, a neuromuscular disease, previous pulmonary tuberculosis, or
another pulmonary disorder, such as chronic bronchitis, were
excluded. Seven patients fulfilling the entry criteria for the study
were referred to the center between May 1983 and July 1983. All
patients gave their informed consent for inclusion in the study.
Table 1—Changes in Hemoglobin, Daytime Arterial Blood Gas Levels, and Overnight Monitoring Between Referral and Reassessment*

<table>
<thead>
<tr>
<th></th>
<th>Initial Referral</th>
<th>Reassessment Admission</th>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>16.3</td>
<td>2.22</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>62.5</td>
<td>16.86</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>53.4</td>
<td>7.30</td>
</tr>
<tr>
<td>Mean overnight SaO₂, %</td>
<td>81</td>
<td>4.2</td>
</tr>
<tr>
<td>Minimum overnight SaO₂, %</td>
<td>54</td>
<td>14.9</td>
</tr>
<tr>
<td>TSaO₂ &lt;90%, %</td>
<td>85</td>
<td>12.3</td>
</tr>
<tr>
<td>DI, dips &gt;4% per hour</td>
<td>12.6</td>
<td>7.91</td>
</tr>
<tr>
<td>Mean overnight PtcCO₂, mm Hg</td>
<td>57.2</td>
<td>9.78</td>
</tr>
</tbody>
</table>

*The mean period between the 2 admissions was 5.08 years; the overnight results on reassessment are those assessed with patient receiving assisted ventilation.

†Significant differences between results at initial referral and the reassessment admission are p<0.05.

‡Significant differences between results at initial referral and the reassessment admission are p<0.01.

Study Design

Assessment at Initial Referral: Information was obtained regarding age of onset of the spinal deformity, duration, and nature of symptoms related to ventilatory failure, and tobacco consumption. A complete physical examination and an ECG were performed. Plain radiographs of the thoracic spine were obtained to determine the level of the kyphosis, and the angulation was estimated using Cobb's method. The hemoglobin concentration and the hematocrit level were measured. PaO₂ and PaCO₂ were measured with the patient at rest breathing room air. The FEV₁ and FVC were measured using a dry spirometer (Vitalograph). Functional residual capacity was measured by helium dilution (P. K. Morgan; Gillingham, United Kingdom) and total lung capacity (TLC) and residual volume were calculated. All volumes were corrected to body temperature, pressure, and saturation and expressed as a percentage of the predicted value calculated on the arm span rather than height to account for the spinal deformity. Maximum mouth pressures were recorded using a calibrated variable reluctance pressure transducer (Si-Plan Electronics Research; Stratford-upon-Avon, England) and expressed as a percentage of predicted values calculated from regression equations according to a previously described method.

The arterial oxyhemoglobin saturation (SaO₂) was recorded continuously overnight using either an ear oximeter (Hewlett Packard; San Diego) or a pulse oximeter (Biox 3700; Ohmeda; Herts, UK) and the transcutaneous partial pressure of CO₂ (PtcCO₂) was recorded using a heated polarographic electrode (TCM3; Radiometrics; Copenhagen). A two-channel recorder, with the paper speed set at 1 mm/min, was used to produce a hard copy. From the paper tracings, the mean and minimum SaO₂ levels, the percentage of time overnight when SaO₂ was less than 90% (TSaO₂ <90%), the desaturation index (DI [number of dips in SaO₂ greater than 4% per hour]), and the mean and minimum PtcCO₂ were calculated and recorded. The indications for the initiation of assisted ventilation and details of the technique of ventilatory support were recorded. Any changes in the method of assisted ventilation during subsequent admissions and the reasons for such changes were recorded.

Reassessment Admission: Between July 1993 and May 1994, the 7 patients were reassessed in detail. All of the investigations performed at initial referral were repeated. In order to observe abnormalities in nocturnal ventilation and to reassess the need for treatment, a controlled withdrawal of assisted ventilation was performed. The patients were monitored on the first night using the ventilator system which they used at home. On the second and third nights, assisted ventilation was withdrawn. On all three nights, SaO₂ and PtcCO₂ were recorded continuously. On the 3rd day, arterial blood gas tensions were measured and on the 4th night polysomnography was performed (Biomedical Monitoring Systems amplifiers and Sleepmaster computer). Chest and abdominal movements were recorded using inductance plethysmography bands (Respi-trace) and nasal-oral airflow was measured with a thermistor. SaO₂ was measured using a pulse oximeter. The EEG was staged using standard criteria; the record was scored for sleep efficiency, and the apnea-hypopnoea index was calculated.

Statistical Analysis

Correlation between the angle of the kyphosis and pulmonary function tests at presentation, the daytime blood gas tensions, hemoglobin levels, and overnight measurements of SaO₂ and PtcCO₂ were examined using Kendall's ranked correlation coefficient. Differences were sought between the results of the laboratory investigations at first referral and those on the first day of the reassessment admission using the Wilcoxon’s matched-pairs signed-rank test. The analysis was performed using the SPSS package (SPSS; Chicago). Probability results of less than 0.05 were accepted as significant.

Results

Initial Referral

Clinical Details at Initial Referral: The 7 patients (4 men) had a mean age at referral of 53 years (SD 7.09). All had had tuberculous osteomyelitis of the spine before the age of 4 years (mean age, 2.2 years; SD 1.1). This had affected the upper thoracic spine in two patients and the midthoracic spine in five. The mean angle of kyphosis at presentation was 133.6° (SD, 14; range, 95° to 135°). The patients had been treated with bedrest and plaster braces. None had received corrective surgery, and only one had received antituberculous chemotherapy.

All patients were ex-smokers (mean number of pack-years was 14; SD 12.6), but none complained of a productive cough or had wheezing on examination. All 7 patients described increasing dyspnea on exertion over a period of at least 4 years. At referral four had dyspnea walking on the flat surface and three had dyspnea at rest. Two patients had morning headaches and all seven described disrupted sleep and daytime somnolence. Six patients were being treated with diuretics, but three of these had persisting peripheral edema.
Table 2—Changes in Pulmonary Function Between Referral and Reassessment

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<th></th>
<th>Initial Referral</th>
<th>Reassessment Admission</th>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>FEV1, % of predicted</td>
<td>19.8</td>
<td>7.47</td>
</tr>
<tr>
<td>FVC, % of predicted</td>
<td>26.8</td>
<td>12.16</td>
</tr>
<tr>
<td>FEV1/FVC ratio, %</td>
<td>0.62</td>
<td>0.10</td>
</tr>
<tr>
<td>TLC, % of predicted</td>
<td>32.0</td>
<td>8.83</td>
</tr>
<tr>
<td>Residual volume, % of predicted</td>
<td>40.2</td>
<td>7.90</td>
</tr>
<tr>
<td>Pimax, % of predicted</td>
<td>72.4</td>
<td>33.6</td>
</tr>
<tr>
<td>Pemax, % of predicted</td>
<td>88.5</td>
<td>26.9</td>
</tr>
</tbody>
</table>

*Significant differences between results at initial referral and the reassessment admission are p < 0.01.
†Significant differences between results at initial referral and the reassessment admission are p < 0.05.
*The percent of predicted values for Pemax at presentation could not be calculated since results for 5 patients were recorded only as more than 100 cm H2O.

Physiologic Measures at Initial Referral: All 7 patients had p pulmonale, right axis deviation, and evidence of right ventricular hypertrophy on an ECG. The results of the arterial blood gas estimations are shown in Table 1. The daytime resting PaCO2 was greater than 50 mm Hg in 6 patients (mean, 53.7 mm Hg; SD 7.30). Results of lung function tests at initial referral are shown in Table 2 and demonstrate a restrictive pattern. In 6 patients TLC and residual volume were both reduced to less than 50% of predicted. Maximal inspiratory mouth pressure (Pimax) was less than 50% of predicted in 2 patients and greater than 85% of predicted in only 2. In 5 patients, maximal expiratory mouth pressure (Pemax) was recorded only as greater than 100 cm H2O and so the percentages of predicted values could not be calculated. However, using a figure of 100 cm H2O for these 5 patients, Pemax was at least 85% of predicted for 6 patients and 36% of predicted for the remaining patient. The complete results of overnight monitoring at initial referral are given in Table 2. Of particular note is the low level of the mean overnight SaO2 (mean, 81%; SD, 4.2), and TSaO2 <90% (mean, 86%; SD, 12.3).

There was no correlation between the angle of kyphosis on the one hand and the hemoglobin concentration, daytime arterial blood gas tensions, the overnight oximetry measures, or maximal mouth pressures on the other hand. There was no overall correlation between the Cobb angles and the measures of lung volume. However, one patient, who did not have any other unusual measurements, did have larger lung volumes despite having the most pronounced kyphosis. If the data of this patient are excluded, there is a strong inverse correlation between the angle of kyphosis and FEV1, TLC, and in particular FVC (r = -0.88; p = 0.02) with each expressed as a percentage of the predicted value (Fig 1).

Progress: Noninvasive nocturnal-assisted ventilation was commenced in all patients. In six patients, the indication was stable ventilatory failure with an elevated daytime PaCO2. The remaining patient had relatively normal daytime blood gas tensions (PaO2, 70 mm Hg; PaCO2, 41.4 mm Hg) but had a mean overnight SaO2 value of 79% and a PtcCO2 level of 53 mm Hg associated with peripheral edema that did not resolve with treatment with diuretics. Cuirass ventilation using customized shells and the Newmarket negative pres-

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21737/ on 06/25/2017)

**Figure 1.** Forced vital capacity (percentage predicted) against angle of kyphosis. There was no overall relationship between vital capacity and the Cobb angle. However, if the outlier (asterisk) is excluded, \( r = -0.88 \) (p = 0.02).
ure pump (Si-Plan Electronics Research; Stratford-upon-Avon, England) was begun for the three patients referred to the center before 1988. The other 4 patients, referred after 1988, began receiving nasal intermittent positive pressure ventilation using standard masks (Respironics; Murrysville, Pa) and the Monnal D positive pressure ventilator in assist-control mode (Taema; Paris, France). None of the patients required additional oxygen therapy at this stage. The dose of diuretics was reduced in all patients taking them, and the diuretics were discontinued completely in two patients.

All patients have been followed up at regular intervals. One patient who had been receiving cuirass ventilation deteriorated after an interval of 29 months with a history suggestive of obstructive sleep apnea and peripheral edema. Overnight monitoring showed a D1 of 28, a mean SaO2 of 83%, and a TSaO2 <90% of 88%, although daytime arterial blood gas values were normal. He was converted to nasal intermittent positive pressure ventilation using a Monnal D ventilator with an improvement in his symptoms and resolution of the peripheral edema. Subsequent overnight monitoring revealed a D1 of 4 and a mean SaO2 of 90%. One other patient who had been receiving cuirass ventilation deteriorated after a period of 34 months with recurrent hypoxemia and peripheral edema (overnight mean SaO2, 81%; TSaO2 <90%, 100%) but no evidence of obstructive sleep apnea. The overnight PtcCO2 showed that he was well ventilated, and daytime PaCO2 while the patient was breathing air was only 43.6 mm Hg. Oxygen therapy was introduced to correct hypoxemia. He died of bronchopneumonia a little more than 9 years after his initial referral. Postmortem examination confirmed right ventricular enlargement and dilated main pulmonary arteries, which was consistent with pulmonary hypertension.

Reassessment Admission

The mean period between the initial referral and the withdrawal studies was 5.08 years (SD 2.50). With the exception of one, all the patients thought that their symptoms were well controlled using nocturnal nasal ventilation with the Monnal D or cuirass ventilation. Six patients reported breathlessness on inclines only and had no morning headaches or daytime somnolence. The remaining patient, who died 3 months after this admission, was breathless on minimal exertion and complained of insomnia. None of the patients had peripheral edema. Improvements were demonstrated in the daytime arterial blood gas values with a statistically significant fall in the PaCO2. The dynamic lung volumes improved in all patients and the differences were statistically significant (p<0.05) although the changes were small. There were no statistically significant changes in maximal mouth pressures. In four patients, however, the Pimax was lower at reassessment, and one patient was too breathless to repeat the measure. Overnight monitoring showed significant improvements in the mean and minimum SaO2 levels and in the TSaO2 <90%. The complete results are given in Tables 1 and 2. The ECG had not altered in any of the patients.

The arterial blood gas levels on the 1st and 3rd days of the reassessment admission were not significantly different. However, there were differences in the overnight oximetry traces on the first and fourth nights, with and without assisted ventilation, respectively. Using Wilcoxon’s matched-pairs sum-ranked test, the mean overnight SaO2 was significantly lower (p=0.04; mean difference, 6.7%) as was the minimum SaO2 (p=0.03; mean difference, 17.3%). The other major finding on the fourth night, when polysomnography was performed, was that 3 of the 7 patients slept very poorly without ventilatory support (sleep efficiency <70%) and one did not sleep at all. As a consequence, the studies were terminated early in these four patients and ventilatory support was restarted during the night. In the 3 patients who spent the whole night without assisted ventilation, the mean sleep efficiency was 83% and mean apnea-hypopnea index was 14.7/h. None of the seven patients had obstructive apneas, but central apneas and hypopneas were demonstrated which were most frequent in rapid eye movement (REM) sleep.

Discussion

We have reported 7 patients with ventilatory failure associated with thoracic kyphosis. This has only been previously documented in a few case reports,7-10 but it may be that the association is more common than is apparent from the literature. Many series report patients with kyphoscoliosis and ventilatory failure.11,12 A true kyphoscoliosis is very rare, and while the term usually is used as a synonym for scoliosis, it is possible that some patients with a genuine kyphosis have been included with the scoliotic subjects in previous series.

This distinction is important because both the etiology of kyphosis and scoliosis and the abnormalities of the respiratory mechanics observed in them are very different. All seven of our patients had had tuberculous osteomyelitis in the thoracic spine. This is recognized to cause a sharp kyphosis or gibbus while scoliosis is usually absent or trivial. The normal thoracic kyphosis has a range of 20° to 40°,13 while in our patients the mean value was 113.6°. In all of the seven subjects, the kyphosis involved the upper or midthoracic vertebrae. Tuberculosis usually infects the vertebrae of the thoracolumbar junction, but it is only when it damages the thoracic vertebrae that important respiratory consequences would be anticipated.

The effects of thoracic scoliosis on respiratory mechanics have been extensively investigated.14 There is a large decrease in the compliance of both the lungs and the chest wall in adults with a Cobb angle of greater than 100°. The respiratory muscles on either side of the asymmetrical chest are affected differently, and the overall influence on maximal mouth pressures appears to depend on the severity of the angulation. In mild scoliosis, with an angle of less than 30°, Pemax has been shown to be reduced more than Pimax,15 while in more severe scoliosis with an angle greater than 60°, Pimax has been found to be considerably reduced while Pemax was preserved.16 With kyphosis, the chest wall may be greatly deformed but remains symmetrical and the changes in orientation of the respiratory muscles are different from those seen with scoliosis. In our patients at referral, Pimax was less than 85% of predicted in all but 2 patients, while Pemax was greater than 85% of predicted for all but 1 patient. It seems likely from our findings that despite the
small lung volumes, diaphragm function is impaired, possibly because it is at a mechanical disadvantage due to its abnormal orientation.

In all of our patients, the kyphosis was apparent by the age of 4 years. This has been a feature of the previously reported cases, and an increased risk of ventilatory failure has been demonstrated in patients with scoliosis of early onset.17 The number of alveoli normally increases until around the age of 8 years but it only does so in response to the mechanical forces generated by the chest wall. The failure of the thoracic cavity to develop, the presence of abnormal respiratory mechanics due to the kyphosis itself, and the ankylosis of the costovertebral joints may have prevented the usual number of alveoli from being formed.18 This would contribute to the restrictive deficit seen in our patients.

Ventilatory failure only developed 43 to 61 years after the kyphosis was noticed, suggesting that it may have been precipitated by changes associated with aging, such as reduction in respiratory drive or chest wall compliance. Loss of respiratory muscle strength probably was a contributory factor because the Pimax was reduced in all but two patients. In other patient groups treated with assisted ventilation, maximal mouth pressures have improved, and it has been argued that this may reflect the relief of respiratory muscle fatigue.19 Because there was no overall improvement in mouth pressures after effective treatment in these patients with kyphosis, we would argue that respiratory muscle fatigue was not an important factor in the development of ventilatory failure.

The patients have been followed up for a mean of 5.7 years (SD, 2.52), and 6 of the 7 patients are still alive. One patient, aged 62, died a little more than 9 years after the initiation of assisted ventilation. Right heart failure (cor pulmonale) has been reported previously in patients with kyphosis,20 and in one series all four patients died.21 Peripherial edema was present in three of our patients at the time of first referral. This resolved in each case once assisted ventilation had been introduced. The electrocardiographic changes of right ventricular hypertrophy did not, however, improve and the patient who died had postmortem evidence of pulmonary hypertension, which was probably a factor in his death.

At reassessment, the mean SaO2 at night improved compared with that at presentation; the values increased from 81 to 92% (p=0.0001). The daytime PaCO2 fell significantly between presentation and the reassessment admission (mean interval, 5.08 years). Similar improvements in daytime blood gas tensions have been demonstrated previously with assisted ventilation in other chest wall disorders, such as scoliosis,11 and after thoracoplasty19 but not in kyphosis. Interestingly, on the fourth night of the reassessment admission in the present study, only three of the seven patients were able to tolerate the whole night off their ventilators. In all patients, there was a deterioration in the mean overnight SaO2 after this short period without assisted ventilation. In a previous study of the withdrawal of ventilatory assistance for a period of 15 days, similar changes in nocturnal gas exchange values were found.22 Our results suggest that even shorter periods of withdrawal may be deleterious.

None of our patients demonstrated obstructive sleep apnea when self-ventilating at night. The records of the three subjects who managed to complete the fourth night of the reassessment admission without assisted ventilation showed central apneas and hypopneas which were most frequent in REM sleep. There have been no previous polysomnographic studies in kyphotic subjects to compare with the findings of the present study. The present observations suggest that either the reduction in biochemical ventilatory drive in REM sleep compared with non-REM sleep and wakefulness or the loss of accessory muscle activity in REM sleep was responsible for these apneas and hypopneas.

These findings suggest that ventilatory failure with kyphosis is associated with an onset of deformity in early childhood and may be more common than has been previously recognized. The cause of the development of ventilatory failure after an interval of over 40 years remains uncertain, but it seems that respiratory muscle weakness is a factor and that abnormalities in nocturnal ventilation precede the deterioration in daytime arterial blood gas values. We have shown that treatment with nocturnal assisted ventilation is well tolerated and can effectively control symptoms and improve physiologic abnormalities in the majority of patients.

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Use of a Bronchial Blocker to Improve Gas Exchange in Respiratory Failure and Differential Lung Disease*

Arthur J. Boujoukos, MD, FCCP; Robert J. Keenan, MD, FCCP

The use of a bronchial blocker to optimize gas exchange in a patient with marked differential lung disease is reported. This technique proved to be a useful alternative in an ICU setting to independent lung ventilation.

(CHEST 1996; 110:1110-11)

Key words: bronchial blocker; gas exchange; respiratory failure

Abbreviations: DLET=double-lumen endobronchial tube; ETT=endotracheal tube; LLL=left lower lobe; PEEP=positive end-expiratory pressure; SLT=single lung transplant

When complicated by a differential lung pathologic condition, respiratory failure may be difficult to manage with conventional approaches. In such cases, knowledge of local ventilation-perfusion ratios and differential compliances within the lungs may be used to tailor effective alternative strategies. The extremes of differential lung pathophysiology are frequently encountered in single lung transplant (SLT) recipients.

SLT recipients with pulmonary hypertension exhibit major ventilation-perfusion mismatch, with 85% or more of the pulmonary perfusion diverted to the allograft by high native pulmonary vascular resistance. In patients with allograft dysfunction, ventilatory management consists of positive end-expiratory pressure (PEEP), sedation, occasionally neuromuscular blockade, and lateral positioning with the native lung down. This last manipulation often is essential to improving ventilation-perfusion matching and oxygenation. In emphysematous SLT recipients, particularly those with allograft dysfunction, differential compliance between the emphysematous native lung and the relatively noncompliant allograft can result in native lung hyperinflation, auto-PEEP, and hypotension. Emphysematic SLT recipients often need independent lung ventilation with low PEEP and a ventilatory rate low enough to prevent auto-PEEP in the native lung while maintaining higher levels of PEEP, smaller tidal volumes, and a higher rate in the stiffer allograft. With independent lung ventilation, however, bronchial anatomy sometimes precludes an effective bronchial seal with a double-lumen endobronchial tube (DLET), and thick pulmonary secretions combined with the small intraluminal size of the DLET make pulmonary toilet extremely difficult.

This case report describes an SLT recipient with both end-stage emphysema and pulmonary hypertension in her remaining native lung, whose severe allograft dysfunction was managed using a bronchial blocker.

Case Report

A 51-year-old woman, 68 kg in body weight and 1.68 m² in body surface area, received a left SLT for end-stage emphysema. Preoperatively, she also had severe pulmonary hypertension with systolic pulmonary artery pressures of 90 mm Hg and a pulmonary vascular resistance index of 1,300 dyne·sec/cm²/m² (normal: 50 to 220 dyne·sec/cm²/m²). After transplantation she had a moderate reperfusion injury requiring independent lung ventilation. A bedside lung perfusion scan performed on the 1st postoperative day revealed that only 5% of the pulmonary blood flow was directed to the hypertensive native lung. By the 4th postoperative day, the PaO₂ was 128 mm Hg on an FIO₂ of 0.5 and PEEP of 5 cm H₂O. The double-lumen tube was removed, and a 8.5 endotracheal tube (ETT) was placed to facilitate clearance of thick bronchial secretions and promote weaning from mechanical ventilation. BAL of an atelectatic left lower lobe (LLL) was required to remove thick brown mucus, and antibiotic therapy was begun. During the next 4 days, arterial oxygenation was less stable, with positioning changes leading to arterial oxygen saturation of 82 to 85%. The patient required full sedation and native lung-down positioning. An arterial blood gas analysis revealed a pH value of 7.31, a PaO₂ level of 69 mm Hg, and PaCO₂ level of 64 mm Hg.

Radiographically, there was no progression of her initial allograft infiltrate. Thick secretions were again aspirated from the LLL, with transient improvement in oxygenation. Attempts to increase PEEP above 7.5 cm H₂O or reduce the inspiration-expiration ratio from 1:4 led to hyperinflation of the native emphysematous right lung and subsequent hypotension and oxygen desaturation. With an empiric diagnosis of allograft rejection, the patient was given the first of three daily doses of methylprednisolone (1,000 mg).

Although independent lung ventilation and selective PEEP to the allograft were considered, the volume and viscosity of the pulmonary secretions were considered to be a major limitation because of the small lumen size of the double-lumen tube required. Because native pulmonary blood flow was thought to be only 5% of total blood flow, it was felt that bronchial blockade could safely limit native lung ventilation and PEEP without significantly increasing shunt. A No. 6 Fogarty catheter was placed through a nasal trumpet and past the vocal cords exterior to the endotracheal tube (ETT) with bronchoscopic guidance. The Fogarty catheter was then positioned into the right mainstem bronchus, and the balloon was inflated with air. Although the balloon was too small to occlude ventilation to the entire lung, it effectively blocked the bronchus intermedius. Oxygenation and CO₂ clearance immediately improved (Table 1). On two occasions, repositioning for a chest radiograph dislodged the bronchial blocker and caused arterial desaturation. This was easily corrected by repositioning the bronchial blocker.