Upper Airway Obstruction following Adult Respiratory Distress Syndrome*
An Analysis of 30 Survivors
C. Gregory Elliott, M.D., F.C.C.P.;† Brad Y. Rasmusson, M.D., F.C.C.P.;§ and Robert O. Crapo, M.D., F.C.C.P.†

To evaluate the effects of current supportive care measures for the adult respiratory distress syndrome (ARDS) upon the upper airway, we studied 30 survivors of ARDS. All patients were interviewed and examined and performed inspiratory and expiratory maximal flow-volume curves more than six months after the onset of ARDS. Three women had developed symptomatic upper airway obstruction due to laryngotracheal stenosis 4 to 12 months after discharge from the hospital. Potential etiologic factors included difficult orotracheal intubation (one) and high tracheal cuff pressures (one). The three survivors who developed laryngotracheal stenoses did not differ from the 27 survivors of ARDS without symptomatic upper airway obstruction with respect to age, duration of tracheal intubation, or maximum level of positive end-expiratory pressure. Each patient with upper airway obstruction required more than one operation for laryngotracheal reconstruction. Although corrective surgery improved airflow, two survivors of ARDS had upper airway obstruction and exertional dyspnea more than five years after the ARDS. We conclude that upper airway obstruction is an important cause of dyspnea and impairment following ARDS. Exertional dyspnea weeks to months following treatment for ARDS suggests the possibility of laryngotracheal stenosis. (Chest 1988; 94:526-30)

The adult respiratory distress syndrome (ARDS) is a clinical syndrome which includes profound arterial hypoxemia, reduced total thoracic compliance, and bilateral diffuse infiltrates on the chest roentgenogram associated with increased vascular permeability.1 Supportive therapy for ARDS includes tracheal intubation and positive-pressure ventilation with positive end-expiratory pressure (PEEP). Patients with ARDS often require prolonged positive-pressure ventilation at high airway pressures.1,2

Injury to the upper airway is a well-recognized complication of tracheal intubation and tracheostomy7-13 and may be particularly likely to occur in patients with ARDS because they require prolonged intubation and ventilation at high airway pressures;7,10,11 however, previous studies of survivors of ARDS rarely mention upper airway obstruction as a sequela of ARDS.2,6,14-16 In the present study, we examined 30 survivors of ARDS for symptomatic and spirometric evidence of upper airway obstruction.

MATERIALS AND METHODS

We reviewed the records of the medical and respiratory intensive care units at LDS Hospital, Salt Lake City, to identify survivors who had ARDS between January 1975 and July 1985. Criteria for the diagnosis of ARDS included the following: severe arterial hypoxemia (ratio of arterial to alveolar oxygen pressure less than 0.2); reduced static total thoracic compliance (less than 50 ml/cm H2O); bilateral diffuse pulmonary infiltrates on the chest roentgenogram; pulmonary capillary wedge pressure of 15 mm Hg or less; and an appropriate clinical setting (eg, sepsis, trauma, aspiration, multiple blood transfusions). We identified 47 survivors of ARDS and attempted to arrange for followup studies. Seventeen survivors were unable to return (n = 12) or were lost to follow-up (n = 5). Those 12 survivors who were unable to return included three who refused to return and nine who were contacted but lived too far away to travel economically. None of the 42 survivors whom we contacted had died during the follow-up period, but we were unable to ascertain survival status for the five survivors who were lost to follow-up.

Follow-up evaluation included completion of an abbreviated respiratory disease questionnaire developed for epidemiologic research by the American Thoracic Society16 and a history and physical examination. Evidence of previous laryngotracheal injury, tracheal intubation, or trauma was specifically sought. Spirometry and measurements of single-breath carbon monoxide diffusing capacity (Db) and helium-dilution total lung capacity (TLCm) were performed according to standard methods.18 Reference values for these tests were those of the Intermountain Thoracic Society.19 Flow-volume loops were performed on a wedge spirometer (Med Science Electronics 750) connected to an X-Y recorder (Hewlett Packard 7045A). Inspiratory and expiratory maximal flow-volume curves were obtained by having the subject perform forced exhalations until reproducible maximal flows and volumes were observed.

In addition, we reviewed the medical records. We recorded the following measures of the severity of pulmonary injury: (1) lowest static thoracic compliance ([exhaled tidal volume — tubing compression volume]/[plateau pressure — PEEP]) measured without an acute reversible change (eg, pneumothorax); (2) initial intrapulmonary shunt fraction measured at a fractional concentration of oxygen in the inspired gas (FIO2) of 1.0; (3) initial mean pulmonary arterial pressure measured at the end of expiration; and (4) the maximum level of PEEP required to maintain an arterial oxygen pressure

*From the Pulmonary Division, Department of Internal Medicine, LDS Hospital, and the Division of Respiratory, Critical Care, and Occupational (Pulmonary) Medicine, Department of Internal Medicine, University of Utah, Salt Lake City.
†Associate Professor of Medicine.
‡Fellow in Pulmonary Medicine.
§Manuscript received October 16; revision accepted February 15. Reprint requests: Dr. Elliott, Pulmonary Division, LDS Hospital, Salt Lake City 84143

526 Upper Airway Obstruction following ARDS (Elliott, Rasmusson, Carpo)
(PaO2) of 55 mm Hg or more and FiO2 of 0.6 or less. We also recorded observations which have previously been associated with laryngotracheal stenoses: (1) the duration of tracheal intubation; (2) the route of intubation; (3) episodes of self-extubation; (4) excessively high tracheal tube cuff pressure (more than 20 mm Hg) recorded at any time during tracheal intubation; and (5) tracheostomy. Records concerning subsequent care related to upper airway injuries were also reviewed. Surgical procedures, complications, and final outcomes were recorded.

Statistical comparisons were made by Wilcoxon's rank-sum test and \( \chi^2 \) analysis.\(^{19} \) Differences were considered significant for \( p < 0.05 \).

**RESULTS**

**Characteristics of Survivors of ARDS**

We evaluated 30 survivors of ARDS between July 1983 and February 1986. The interval between the onset of ARDS and the follow-up evaluation ranged from six months to eight years (mean, four years). The mean age at onset of ARDS was 28 years (range, 13 to 62 yrs). Physiologic characteristics (mean ± SEM) measured during supportive care for ARDS included the following: (1) lowest static thoracic compliance, 23±2 ml/cmH2O; (2) initial intrapulmonary shunt fraction, 0.32±0.03; (3) the initial mean pulmonary arterial pressure, 27±1 mm Hg; and (4) the maximum PEEP required to maintain PaO2≥55 mm Hg and FiO2≤0.6=21±1 cmH2O. The duration of tracheal intubation ranged from 3 to 35 days (mean, 16±3 days).

None of the 30 survivors of ARDS had tracheal trauma or cutaneous burns or had inhaled a caustic gas, and none had disease of the upper airway prior to developing ARDS. One of the survivors had been intubated two years earlier for a routine cholecystectomy, but she did not have laryngotracheal stenosis after ARDS. One survivor who developed laryngeal stenosis had been intubated uneventfully during surgical repair of a fractured femur six years before developing ARDS. Initial intubations were performed by a variety of medical personnel including anesthesiologists, pulmonary internists, and hospital physicians. Fifteen (50 percent) required one or more reintubations because of cuff leak (n=7) or self-extubation (n=7) or because excessive cuff pressures (>20 mm Hg) were necessary to prevent the loss of tidal volume or PEEP during ventilatory support (n=2). Seven (23 percent) of 30 underwent tracheostomy 1 to 25 days after the initial intubation.

More than six months after the onset of ARDS, seven of 30 survivors noted dyspnea when walking with others of equal age (Table 1). Three of these seven had surgical corrections for laryngotracheal stenoses. Three of the remaining four with exertional dyspnea had reductions of forced vital capacity (FVC), TLC, and Dsb which suggested residual restrictive pulmonary disease but had no evidence of central airway obstruction. Spirometric data, pulmonary volumes, and the Dsb of the fourth symptomatic survivor were normal.

**Survivors of ARDS with Laryngotracheal Stenosis**

Three survivors developed symptomatic laryngotracheal stenoses accompanied by physiologic evidence of upper airway obstruction following orotracheal intubation (Table 2).\(^{20} \) These three patients had not undergone tracheostomy during the time they were intubated for ARDS or during the interval between extubation and presentation with upper airway obstruction. Laryngeal and tracheal endoscopic examinations identified the locus of obstruction in each case. The first patient presented with stenosis of the larynx between the vocal cord processes of the two arytenoid cartilages and required emergent tracheostomy. The second patient developed fibrous adhesions of the subglottic larynx and the superior trachea. This patient had two flow-volume curves performed which demonstrated progressive upper airway obstruction (Fig 1) before undergoing a tracheostomy. The third patient also required emergent tracheostomy because of ankylosis of the cricoarytenoid joints and a granuloma of the right arytenoid. The remaining 27 survivors of ARDS performed maximal forced exhalations and inhalations (flow-volume curves) which did not suggest

**Table 1—Anthropomorphic Data and Pulmonary Function of Seven Survivors with Exertional Dyspnea More than Six Months after ARDS***

<table>
<thead>
<tr>
<th>Patient, Sex, Age (yr)</th>
<th>Tracheal-laryngeal Stenosis</th>
<th>Smoking History (pack-years)</th>
<th>Height (cm)</th>
<th>FVC, L†</th>
<th>FEV/FVC, percent</th>
<th>TLC, L†</th>
<th>Dsb, ml/mm Hg/min</th>
<th>P(A-a)O2, mm Hg‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, F, 45</td>
<td>+</td>
<td>0</td>
<td>164</td>
<td>2.73 (78)</td>
<td>82 (100)</td>
<td>4.63 (90)</td>
<td>25.7 (91)</td>
<td>20</td>
</tr>
<tr>
<td>2, F, 18</td>
<td>+</td>
<td>0</td>
<td>166</td>
<td>3.39 (81)</td>
<td>80 (91)</td>
<td>3.84 (73)</td>
<td>35.9 (108)</td>
<td>23</td>
</tr>
<tr>
<td>3, F, 28</td>
<td>+</td>
<td>0</td>
<td>168</td>
<td>3.92 (97)</td>
<td>73 (86)</td>
<td>5.23 (97)</td>
<td>27.2 (85)</td>
<td>12</td>
</tr>
<tr>
<td>4, F, 37</td>
<td>−</td>
<td>37</td>
<td>164</td>
<td>3.09 (84)</td>
<td>57 (103)</td>
<td>4.44 (86)</td>
<td>19.5 (66)</td>
<td>16</td>
</tr>
<tr>
<td>5, M, 19</td>
<td>−</td>
<td>0</td>
<td>172</td>
<td>3.93 (75)</td>
<td>80 (94)</td>
<td>5.48 (86)</td>
<td>31.9 (79)</td>
<td>8</td>
</tr>
<tr>
<td>6, F, 47</td>
<td>−</td>
<td>50</td>
<td>163</td>
<td>3.14 (92)</td>
<td>85 (104)</td>
<td>4.84 (95)</td>
<td>24.5 (88)</td>
<td>15</td>
</tr>
<tr>
<td>7, F, 54</td>
<td>−</td>
<td>0</td>
<td>152</td>
<td>1.06 (39)</td>
<td>67 (81)</td>
<td>2.24 (51)</td>
<td>8.9 (38)</td>
<td>27</td>
</tr>
</tbody>
</table>

*Values in parentheses indicate percent predicted.
†Body temperature and pressure, saturated.
‡Alveolar-arterial oxygen pressure difference.
upper airway obstruction. Tracheal tomograms and laryngoscopy were not routinely performed for these 27 survivors.

Survivors of ARDS with laryngotracheal stenosis did not differ from those without this complication with respect to age, duration of intubation, or maximum level of PEEP (Table 3). The risk of developing upper airway obstruction appeared greater for women (3/16) than for men (0/14), but this difference was not statistically significant (p = 0.27). Similarly, each of the three patients who subsequently developed laryngotracheal stenoses had been supported with orotracheal tubes without tracheostomy for periods ranging from 10 to 24 days; however, χ² analysis of oral vs nasotracheal intubations and the presence or absence of symptomatic upper airway obstruction was not statistically significant (p>0.23). Examination of potential etiologic factors in the development of upper airway obstruction revealed that the patient with a supraglottic web and fibrous adhesions of the superior trachea had required tracheal tube cuff pressures in excess of 35 cm H₂O to maintain ventilatory support. Intubation of the patient who was subsequently found to have arytenoid of the cricoarytenoid joints was prolonged and difficult.

**DISCUSSION**

A number of investigators have examined the prognosis for recovery following ARDS. These reports have documented recovery to normal or nearly normal pulmonary function within six to nine months after ARDS and have suggested that disability is uncom-

---

**Table 2—Complications Experienced by Survivors of ARDS with Symptomatic Upper Airway Obstruction**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Presenting Symptom</th>
<th>Pathology</th>
<th>Therapy</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Dyspnea and hoarseness 5 mo after discharge</td>
<td>Posterior commissure stenosis of larynx (fibrous adhesions)</td>
<td>Excision of granulation tissue from vocal cord processes of two arytenoid cartilages; Teflon injection of vocal cord</td>
<td>Preop, 3-mm orifice; postop, 7-mm orifice*</td>
</tr>
<tr>
<td>2</td>
<td>Dyspnea and stridor 4 mo after discharge</td>
<td>Subglottic and superior tracheal stenosis (fibrous adhesions)</td>
<td>Excision of granulation tissue from subglottic larynx and superior trachea</td>
<td>Preop, 3-mm orifice*; postop, 6-mm orifice*</td>
</tr>
<tr>
<td>3</td>
<td>Dyspnea and hoarseness 12 mo after discharge</td>
<td>Posterior commissure stenosis of larynx with ankylosis of cricoarytenoid joints and laryngeal granuloma</td>
<td>Excision of granulation tissue and granuloma from right arytenoid</td>
<td>Postop, 5-mm orifice*</td>
</tr>
</tbody>
</table>

*Tracheal diameter estimated from flow-volume analysis according to Gamsu.¹⁰
mon.14,13 Our prior studies14,21 have supported the conclusion that severe pulmonary dysfunction is rare after ARDS. The present study suggests that upper airway obstruction is an important late complication of current supportive measures for ARDS. Recognition of upper airway obstruction following ARDS is important, since dyspnea may be erroneously attributed to residual pulmonary injury and because surgical correction is possible.10,13 Furthermore, upper airway obstruction can be life-threatening.

The pathology of upper airway obstruction following ARDS was not specific. We encountered upper airway lesions which have been described previously following prolonged tracheal intubation.8,9 Laryngeal granulomas and polyps complicate brief intubations26 and occur more commonly after prolonged intubation.23,24 They usually occur weeks after intubation and require surgical excision.9 Laryngeal fibrosis leads to airway obstruction by narrowing the subglottic lumen or by immobilizing the vocal cords.8,9 As illustrated by two patients in the present series, complete surgical correction may not be possible. Tracheal stenoses usually occur where the tracheal tube cuff compresses the tracheal wall. We did not identify tracheal stenoses attributable to the tracheal tube cuff or tracheostomy stoma injuries. We suspect that these lesions were present in some patients but were not severe enough to cause clinical symptoms or spirometric abnormalities.

We identified symptomatic upper airway obstruction in 10 percent (3) of the 30 survivors of ARDS who returned for evaluation. Because the present study did not include routine tracheal tomograms or laryngoscopic examination, the incidence of residual laryngotracheal pathology was probably underestimated. Stauffer et al,7 in a prospective study of 150 adults requiring more than 24 hours of tracheal intubation for respiratory failure of various causes, were able to evaluate 44 survivors. Using tomograms of the larynx and trachea, these investigators24 identified tracheal stenosis in 16 (36 percent), but the tracheal diameter was reduced by more than 50 percent in only two patients (5 percent). Previous work suggests that tracheal tomograms and laryngoscopic examinations have a greater sensitivity for the detection of laryngotracheal lesions than history, physical examination, or flow-volume curve analysis.7,11,24,25 Andrew and Pearson25 suggest that at least a 50 percent decrease in tracheal diameter must occur before dyspnea and stridor are detectable. Therefore, tracheal tomograms and laryngoscopic examinations of our patients likely would have identified additional laryngotracheal sequelae of supportive care for ARDS. Our observation of symptomatic upper airway obstruction in 10 percent of the survivors of ARDS exceeds that reported for patients who required prolonged tracheal intubation for respiratory failure of various causes. Kastanos et al24 reported positive associations between established laryngeal lesions and the use of PEEP. The present study cannot discern whether ARDS or associated phenomena (use of higher tracheal tube cuff pressures in order to apply PEEP) increase the risk for laryngotracheal stenosis.

The three survivors of ARDS with symptomatic upper airway obstruction presented with dyspnea several months after discharge from the hospital. In one patient, we demonstrated progressive tracheal obstruction during the fifth month after extubation. The delayed onset and the progressive nature of upper airway obstruction contrasts sharply with the natural history of recovery after ARDS. Dyspnea related to pulmonary injury decreases after ARDS as vital capacity, TLC, and Ds increase.14 In the present series, one patient with persistent pulmonary fibrosis noted improvement in dyspnea during the first six months after ARDS. Thus, we believe a survivor of ARDS who describes worsening exertional dyspnea several weeks or months following discharge from the hospital should be evaluated for laryngotracheal stenosis.

A number of factors have been associated with upper airway injuries following prolonged tracheal intubation. Most investigators have identified tracheal stenoses where the tracheal tube's cuff sealed against the tracheal mucosa, suggesting that trauma and ischemia lead to injury and stenosis.26-28 Colice et al29 have proposed that tracheal tubes and inhalation injuries associated with cutaneous burns may act synergistically to produce tracheal stenosis. Although ARDS may complicate burns or inhalation injuries, our three patients who developed upper airway obstruction after ARDS had not suffered inhalation injuries or burns. In the present series, the three patients who developed laryngotracheal stenosis following ARDS were women who received positive-

### Table 3—Characteristics of Survivors of ARDS*

<table>
<thead>
<tr>
<th>Data</th>
<th>n</th>
<th>Age at Onset of ARDS, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Duration of Tracheal Intubation, days</th>
<th>Maximum PEEP, cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>No laryngotracheal obstruction</td>
<td>27</td>
<td>28 ± 2</td>
<td>169 ± 3</td>
<td>70 ± 3</td>
<td>16 ± 3</td>
<td>21 ± 2</td>
</tr>
<tr>
<td>Laryngotracheal obstruction</td>
<td>3</td>
<td>24 ± 7</td>
<td>168 ± 1</td>
<td>88 ± 11</td>
<td>15 ± 4</td>
<td>20 ± 10</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>28 ± 2</td>
<td>169 ± 1</td>
<td>72 ± 3</td>
<td>16 ± 3</td>
<td>21 ± 1</td>
</tr>
</tbody>
</table>

*Table data are means ± SEM. p>0.05 for all comparisons of survivors with symptomatic upper-airway obstruction and those without symptomatic laryngotracheal obstruction.
pressure ventilation through an orotracheal tube. The involvement of the vocal cords and the subglottic larynx in our patients may relate to prolonged orotracheal intubations. Stauffer et al. found a highly significant association between ulceration of the posterior glottis and oral intubation. The retrospective collection of data related to airway management limited our analysis of associated causes for upper airway obstruction in these survivors of ARDS.

In summary, we conclude that upper airway obstruction is an important cause of dyspnea and impairment following ARDS. This observation deserves emphasis because correct diagnosis and treatment can provide symptomatic and functional improvement. In our experience, progressive dyspnea during the first year after ARDS is more likely due to laryngotracheal obstruction than to residual pulmonary dysfunction. For this reason, evaluations of symptomatic survivors of ARDS should include maximal inspiratory and expiratory flow-volume measurements and endoscopic examination when patterns suggesting upper airway obstruction are observed.

ACKNOWLEDGMENT: We thank Ms. Debbi Crews and Ms. Harriet May for technical assistance and Mr. Keith Green for manuscript preparation.

REFERENCES

6 Buchser E, Leuenberger PH, Chiodero R, Perret CL, Freeman J. Reduced pulmonary capillary blood volume as a long-term sequel of ARDS. Chest 1985; 87:608-11
10 Geffin B, Grillo HC, Cooper JD, Pontoppidan H. Stenosis following tracheostomy for respiratory care. JAMA 1971; 216: 1984-88
11 Dane TEB, King EG. A prospective study of complications after tracheostomy for assisted ventilation. Chest 1975; 67:398-404
15 Alberts WM, Priest GR, Moser KM. The outlook for survivors of ARDS. Chest 1983; 84:272-74
17 Ferris BG. Epidemiology standardization project. Am Rev Respir Dis 1978; 118:10-36
28 Gibson P. Etiology and repair of tracheal stenosis following tracheostomy and intermittent positive pressure respiration. Thorax 1967; 22:1-6