Table II. *Echocardiographic findings*

	<i>Benign effusions</i>		<i>Malignant effusions*</i>	
	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>
Right atrial collapse	6/12	(50)	9/12	(75)
Right ventricular collapse	4/12	(33)	8/12	(67)
Left atrial collapse	1/12	(8)	4/12	(33)
Cardiac tamponade†	2/12	(17)	7/12	(58)
Dilated inferior vena cava	2/12	(17)	1/12	(8)
Fibrin	10/12	(83)	7/13	(54)
Loculated	4/12	(33)	4/13	(31)
Pericardial masses	5/12	(42)	2/13	(15)

*Chamber collapse was assessed in only 12 of 13 patients with malignant effusion.

†Moderate to severe right atrial, right ventricular, or left atrial collapse.

malignant effusions and three with benign effusions: none of the cultures were positive for bacterial or fungal infection.

Echocardiographic findings and their distribution between the benign and malignant effusions are listed in Table II. In addition, the average volume was estimated to be 287 ml (median 250 ml) in the 19 patients for whom complete data were available. Nine of the malignant effusions (69%) were classified as large, as were six of the benign effusions (50%). Right atrial collapse was severe in one patient with malignant effusion, moderate in six patients (malignant, $n = 5$; benign, $n = 1$), mild in eight (malignant, $n = 3$; benign, $n = 5$), and not present in nine (malignant, $n = 3$; benign, $n = 6$). Right ventricular collapse was severe in one patient with a malignant effusion, moderate in four (malignant, $n = 2$; benign, $n = 2$), mild in seven (malignant, $n = 5$; benign, $n = 2$), and not present in 12 (malignant, $n = 4$; benign, $n = 8$). Left atrial collapse was moderate in one patient with benign

effusion, mild in four with malignant effusion, and not present in 18 (malignant, $n = 7$; benign, $n = 11$). Left ventricular function was normal in 22 patients, hypocontractile in one patient with benign effusion, and could not be characterized in two. Overall, cardiac tamponade, as evidenced by moderate to severe collapse of one or more chambers, was more common in patients with malignant effusions than benign effusions ($\chi^2 = 4.44$, $0.02 < p < 0.05$, $n = 24$). The median survival of patients with benign effusions was 219 days (range 8 days to 3.5 years) and for patients with malignant effusions 81 days (range 5 days to 3.8 years); two patients with benign effusions were alive at the end of the follow-up period.

Overall, echocardiographic evidence for the presence or absence of fibrin bands, loculations, pericardial masses, or inferior vena caval dilatation could not be correlated with the presence of malignancy, although moderate to severe chamber collapse did correlate with the diagnosis of malignant pericardial effusion. Pericardial fluid cytology and pericardial sac pathology are complementary tests, with the latter often informative when the former is negative; thus obtaining both pericardial tissue and fluid will increase the frequency of detection of intrapericardial malignant disease.

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SUCCESSFUL ESOPHAGEAL TRACHEOBRONCHOPLASTY FOR COMBINED TRACHEAL AND BRONCHIAL TRAUMATIC RUPTURE

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A mother fell down a flight of stairs while carrying her 5-year-old son in her arms. As she fell, she tightened her grip around the child's chest, leading to a blunt chest trauma with reflex closure of the glottis associated with trauma to the head. A medical emergency unit found the boy unconscious with subcutaneous emphysema of the neck and both hemithoraces. He was intubated without sedation with a No. 5 cuffed nasotracheal tube, and positive-pressure ventilation was initiated.

On admission to the hospital 1 hour later, the child was conscious and his respiratory status remained stable. A

Fig. 1. Thoracic computed tomographic scan showing the esophagus protruding into the tracheal lumen (arrow).

chest radiograph showed a partial bilateral pneumothorax that was larger on the right side, with extensive subcutaneous and mediastinal emphysema. A thoracic computed tomographic scan showed that the esophagus was protruding into the tracheal lumen through its posterior wall (Fig. 1). Examination with a flexible bronchoscope revealed a 4 cm posterior longitudinal tear from the trachea to the right main bronchus. Despite bilateral pleural drainage, the child's respiratory status deteriorated rapidly and he was transferred to the operating room with acute respiratory failure. Selective ventilation of the left lung was not possible because the endotracheal tube could not be advanced far enough beyond the laceration into the left main-stem bronchus. A right lateral thoracotomy was quickly performed. As the posterior mediastinal pleura was progressively opened, detailed inspection of the injury revealed an 11 cm burstlike fracture with a major defect of the membranous pars from the trachea to the right main bronchus and the bronchus intermedius (Fig. 2, A). Gas exchange seriously deteriorated because of the extent of the air leak. An attempt to perform high-frequency jet ventilation with the tip of the gas inflow catheter advanced distal to the rupture failed. The acute condition of the patient and the length of the tracheobronchial rupture necessitated that an esophageal tracheobronchoplasty be performed to reconstruct the posterior wall of the trachea. The right edge of the esophagus was sutured to the right edge of the wound from the trachea to the bronchus intermedius with interrupted 5-0 absorbable monofilament sutures (Fig. 2, B). An immediate air seal was obtained. The tracheal tube was positioned above the superior edge of the wound before chest closure. Further recovery required bronchoscopic toilet each day to keep the airway clear of secretions.

The child was extubated on day 2, the thoracic drains were removed on day 6, and he was discharged on day 10. A follow-up bronchoscopic examination at 3 months

Fig. 2. A, Rupture of the membranous pars with a large defect from the trachea to the right main-stem bronchus and the bronchus intermedius. B, The right side of the rupture was sutured to the right side of the esophagus with interrupted monofilament absorbable suture.

showed that the tracheobronchial tree looked normal. The child was doing well without any symptoms 1 year after the operation.

Rupture of the tracheobronchial tree is an uncommon complication of blunt chest trauma. In our young patient, the reflex closure of the glottis together with thoracic compression produced a rapid increase in intraluminal pressure. This injury occurred when the child's mother tightened her grip around him, illustrating the fact that extreme violence is not essential for such burstlike lesions.² Overinflation of the tracheal cuff when the accident occurred may have contributed to the extent of the injury. Moreover, the extensive tearing of the membranous pars can be explained by further dissection with the aid of positive ventilatory support.

When a major tracheal or bronchial injury is suspected, bronchoscopy should be performed to establish the precise location of the injury. Nevertheless, this procedure can also lead to an underestimation of the extent of injury.³ Computed tomographic scans may also provide additional information regarding the severity of the injury, such as in the case described here where the esophagus was found to be protruding into the tracheal lumen.

Anesthetic management of the ruptured airway can be hazardous in pediatric patients. In the case of rupture of the carina or main bronchus, double-lumen tubes are not available in pediatric sizes, and tubes with bronchial blockers could exacerbate the injury. Consequently, selective intubation with the aid of bronchoscopic guidance with a long single-lumen tracheal tube is the best technique to achieve appropriate ventilation.⁴ Intubation of the left main bronchus through the operative field is another possibility. None of these maneuvers were possible in the case of the small child described here, however, whose condition was extremely unstable. An intraoperative air seal was the sole procedure available to us to

restore proper ventilation. Tracheoplasty with the esophageal wall was initially described to treat patients with congenital tracheal stenosis. Niwa and associates⁵ first reported the delayed treatment of an iatrogenic tracheobronchial injury with this method.

The operative technique we used differed from the one described by Niwa, who sutured the left and right edges of the trachea to the esophagus.

This is, to our knowledge, the first report of such a technique being successfully used in an emergency to repair a tracheobronchial rupture. We used this procedure because it was the most expeditious way to achieve a perfect air seal. In addition, the abundant blood supply to the esophageal wall makes it superior to an isolated pericardial graft to repair such a large defect. The esophageal wall was completely epithelialized 3 months after the procedure was performed, and the absence of tracheobronchial stenosis by granulation tissue confirms that this technique can be used safely in young children.

In conclusion, esophageal tracheobronchoplasty is a simple and efficient method to obtain immediate and complete reconstruction of the tracheal wall in cases of

extended burstlike trauma with further ad integrum recovery of both airway and esophageal functions.

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LONG-TERM SURVIVAL AFTER RESECTION OF A MEDIASTINAL METASTASIS FROM A RENAL CELL CARCINOMA

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Twenty-five percent to 30% of patients with a renal cell carcinoma have a metastasis by the time the renal cell carcinoma is diagnosed, and 30% to 50% of these patients subsequently have pulmonary metastases.^{1,2} Pulmonary metastases are common in patients with renal cell carcinoma and usually consist of multiple nodules of varying sizes that develop in both lung fields. In contrast, mediastinal adenopathy but no pulmonary parenchymal disease is a rare manifestation of intrathoracic involvement. Herein we describe the case of a patient with a mediastinal metastasis of a renal cell carcinoma. A mediastinal lymph node resection was performed, and 6 years after the operation the patient remains alive.

Clinical summary. Twelve months before admission to our facility, our patient, a 50-year-old man, had undergone a left nephrectomy. He appeared to be doing well after the operation until a routine follow-up chest radiograph revealed a right paratracheal adenopathy (Fig. 1).

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Chest computed tomography (CT) showed paratracheal adenopathy (Fig. 2) but no parenchymal metastases. Bronchoscopic examination revealed nothing abnormal and cytologic studies of sputum and bronchial washings revealed no contributory findings. Other tests, which included abdominal CT scans, brain CT scans, gallium scintigraphy, and bone scintigraphy, showed no evidence of any other tumors.

Thus thoracotomy was performed and the mass, located between the trachea and the superior vena cava, was excised. The tumor, measuring 58 × 55 × 35 mm, was encapsulated and easily resected, and a subsequent pathologic examination confirmed that it was a metastatic, lymphoid tissue renal cell carcinoma. The patient was given no postoperative therapy and to date, 6 years after the operation, has had no recurrence of tumor.

Comments. Pulmonary metastasis of a renal cell carcinoma itself is an initial feature of the disease in only 2% of cases and in such cases usually appears as multiple nodules in both lung fields.² Patients with renal cell carcinoma who have no parenchymal abnormalities but do have a hilar or paratracheal adenopathy, indicating intrathoracic involvement of the renal cell carcinoma, are rarely encountered. In a series of 1451 patients, only 75 (5%) were found to have a mediastinal adenopathy, whereas more than 90% had pulmonary parenchymal metastases and no isolated lymph node involvement in the chest cavity.³ The 5-year survival of patients with unresected metastatic renal cell carcinoma is only 2.7%.⁴ As for treatment of patients with metastatic renal cell carci-