days after BAE with alcohol from massive hemoptysis secondary to a fistula between the left pulmonary artery and the adjacent necrotic origin of the left upper lobe bronchus. In another case, BAE with an absorbable gelatin sponge (Gelfoam) resulted in a fistula between the esophagus and the left main-stem bronchus, with a very localized necrosis of the posterior wall of the bronchus and a larger ischemic area in the esophagus. Surgical repair with bronchial and esophageal sutures, however, appeared successful in this case. A zone of necrosis on a right main-stem bronchus was also reported after absorbable gelatin sponge embolization with a slow resolution within a few weeks. Finally, an asymptomatic 3 × 4-mm area of infarction of the mucosa of the left main-stem bronchus was described after embolization with a 10 percent solution of sodium chloride, two weeks later, however, the lesion was no longer detectable. Whatever the true incidence of severe ischemic lesions induced by BAE, the predisposing factors for such complications remain unclear. In our observation, the probable vascularization of the roof of the main-stem bronchus by the embolized artery trunk, together with the moderate hypertrophy of this vessel and the absence of evident anastomoses with other arteries perhaps constitute risk factors for BAE-induced ischemia. Furthermore, although ischemia has been described with virtually all embolization agents, the nonresorbable character of isobutyl-2-cyanoacrylate might be incriminated in the failure of postischemic healing mechanisms.

To our knowledge, only five cases of balloon-catheter dilatation of bronchial stenosis in adults have been reported in the literature. Partial or complete success was obtained in all cases, with times of follow-up ranging from a few weeks to 14 months. In two cases, however, laser photoresection was associated with balloon dilatation and the respective effects of each procedure were not clearly determined. In our patient, a small degree of dilatation was obtained with the balloon catheter alone, as proved by immediate postdilatation bronchography, diminished wheezing, and subsequent bronchofiberscopic controls. This technical success, however, was not beneficial to the patient because major distal bronchial lesions were evident already and left pneumonectomy could not be avoided.

We conclude that clinicians and radiologists must be aware of the possibility of severe late bronchial sequelae following BAE and that more information is needed regarding the pathophysiology and, consequently, the prevention of this rare but severe complication.

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Pulmonary Edema Associated with Electrical Injury*

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The occurrence of cardiogenic pulmonary edema following alternating current electrical injury has not been reported. A patient developing severe pulmonary edema immediately following an electrical injury-induced episode of ventricular fibrillation is described. Evidence that the etiology of the pulmonary edema was cardiogenic is derived from both hemodynamic data and the calculation of the pulmonary edema fluid to serum colloid osmotic pressure ratio.

(Chest 1990; 97:1248-50)

Electrical injury in power line workers and the subsequent financial support from utility companies were instrumental in early modern resuscitation research and the development of defibrillators for clinical use. The application of direct current for the conversion of atrial fibrillation has been associated with pulmonary edema, but the mechanism of edema formation in these cases remains unknown. The cardiopulmonary sequelae of alternating current electrical injury have received little attention in the medical literature. The occurrence of pulmonary edema and the status of clinical cardiac function after injury have not been reported. A review of the treatment of lightning injury describes a patient who developed pulmonary edema, but provides no data supporting the etiology as cardiogenic or noncardiogenic. It is plausible that the

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Pulmonary Edema Associated with Electrical Injury (Schein et al)
mechanism of edema formation might be either cardiogenic or noncardiogenic: noncardiogenic as a consequence of either trauma or burns, or cardiogenic as a consequence of direct myocardial injury. We report a case in which a patient developed cardiogenic pulmonary edema following an alternating current electrical injury.

**Case Report**

A 57-year-old white man collapsed while working on an electrical panel (approximately 480 volts). After they were unable to detect a pulse, co-workers immediately started artificial ventilation and chest compression. Approximately three minutes later, a rescue squad determined that the patient was in ventricular fibrillation and defibrillated the patient with one 300 joule shock. The patient then developed ventricular tachycardia which converted to sinus rhythm with the administration of 100 mg of lidocaine. The patient was intubated, started on a lidocaine infusion at 2 mg/min and was transported to the hospital. The patient received approximately 350 ml of 5 percent dextrose solution before he reached the hospital but no further therapy.

In the emergency department, the patient was in normal sinus rhythm at a rate of 90 beats per minute, his blood pressure was 180/120 mm Hg, and he had a spontaneous respiratory rate of 30 breaths per minute. He did not respond to verbal commands, but moved spontaneously and in response to noxious stimuli. His physical examination was notable primarily for copious amounts of pink, frothy pulmonary edema fluid draining from his endotracheal tube and bilateral wheezes, rhonchi and rales. Heart sounds were obscured by the lung sounds, but no murmurs, rubs or gallops were heard. There was no jugular venous distension or peripheral edema. The patient’s medical history included a right lower lobe lobectomy several years prior to admission, left hemorrhhaphy, and mild asthma. The patient had no history or symptoms of cardiac disease.

Initial serum electrolyte and CPK values were within normal limits. Chest roentgenogram showed opacification of the right lung and interstitial edema on the left. The initial electrocardiogram showed a normal sinus rhythm and 2 mm ST segment elevations in the lateral precordial leads. Arterial blood gas values, on arrival at the emergency room with the patient receiving manual ventilation and 100 percent FIO2, were pH: 7.09, PO2: 86 mm Hg, PCO2: 45 mm Hg.

The patient was admitted to the intensive care unit, where mechanical ventilation was started and a pulmonary artery catheter was placed. The initial pulmonary artery occlusion pressure was 16 mm Hg, the cardiac index was 2.6 L/min/m2, the Po2 was 31 mm Hg, 62 percent saturation, and the systemic vascular resistance was 1408 dynes-cm-5 on 5 cm H2O PEEP. Over the next six hours, the blood pressure was maintained at 100 to 120/50 to 70 mm Hg on therapy with 5 to 10 μg/kg/min of dopamine. The pulmonary artery occlusion pressures ranged from 16 to 22 mm Hg and the cardiac index fell to 2.2 L/min/m2, while PEEP remained at 5 cm H2O. Samples of both pulmonary edema fluid and serum obtained on admission were analyzed for colloid osmotic pressure and showed values of 8.7 and 18.6 mm Hg, respectively, and a pulmonary edema fluid to serum ratio of 0.47. Ventilatory support consisted of reducing the FIO2 to 50 percent, while maintaining an arterial oxygen saturation of at least 90 percent by incremental increases of PEEP. This required a maximum PEEP of 12 cm H2O.

Over the following 72 hours, the chest x-ray film showed a decrease in interstitial edema, the cardiac index increased to 4.3 L/min/m2; and the patient was successfully weaned from mechanical ventilation. The ECG showed T wave inversions in leads 1, aVL, and V5-V6, that persisted throughout the remainder of the hospital course. Serum creatine phosphokinase value increased to a peak of 7,500 U/L at 96 hours, but fractionation showed 100 percent mm bands. Over this time, the patient developed typical lesions of first degree burns over both arms and shoulders. On the tenth hospital day, the patient was transferred to another facility where he made an uneventful recovery. On the 17th day after his injury, an echocardiogram showed normal left atrial and ventricular cavity sizes and good ventricular function with minimal hypokinesia. A rest MUGA scan obtained at the same time showed an ejection fraction of 49 percent and an area of relative hypokinesia involving the lateral wall of the left ventricle. Following discharge, the patient remained without symptoms or signs of cardiac disease. An ECG obtained two years following discharge demonstrated a return to the preinjury tracing.

**Discussion**

The patient described above sustained a brief period of ventricular fibrillation initiated by an alternating current shock. A cardiogenic etiology of the pulmonary edema is supported by several lines of clinical evidence. The pulmonary artery catheter data obtained shortly after admission revealed an elevated pulmonary artery occlusion pressure, reduced cardiac index and low oxygen saturation of mixed venous blood. Analysis of the pulmonary edema fluid to serum colloid osmotic pressure ratio was consistent with a small amount of protein in the pulmonary edema fluid relative to the serum, supporting the diagnosis of cardiogenic pulmonary edema. 44 In addition, the absence of risk factors for inhalation injury and the lack of significant volume resuscitation suggest that neither thermal lung injury nor volume overload were factors.

The proximate cause of the cardiac dysfunction in this patient cannot be determined unequivocally. Possibilities include preexisting cardiac failure, acute myocardial infarction, and ventricular dysfunction related to either electrical injury or prolonged ischemia during resuscitation. The patient had a normal ECC and no history suggestive of ischemic heart disease or congestive heart failure prior to his injury. Over the two years following his injury, his ECG returned to baseline and the patient remained without symptoms or signs referable to cardiac disease. While these factors cannot rule out significant ischemic cardiac disease, they do argue against its presence. However, while cardiac enzyme determinations made during his hospital stay were negative, ECGs were consistent with a pattern of ischemia. The echocardiogram and MUGA scan performed approximately two weeks after injury were both indicative of adequate overall ventricular function but also of mild regional hypokinesia.

Was this patient's cardiac dysfunction related to the electrical injury per se or a consequence of the cardiac arrest induced by the electrical injury? That he received immediate bystander cardiopulmonary resuscitation and was promptly defibrillated with the rapid return of an adequate blood pressure argue against prolonged ischemia as the predominant cause of cardiac dysfunction. The possibility that the defibrillation itself was responsible for this patient's cardiac dysfunction is unlikely. Pulmonary edema and cardiac depression have not been reported as clinical complications of electrical defibrillation of ventricular fibrillation, although experimental models using the direct application of electrodes to epicardium or endocardium have produced both depression of myocardial contractility and abnormalities of cardiac blood flow. 45 However, higher cumulative energies or more closely spaced
Repeated shocks were required to produce these changes. Similarly, a study employing technetium-99m stannous pyrophosphate scanning and pathologic examination in dogs exposed to direct current shocks found little evidence of myocardial injury at low energy levels, but substantial evidence of necrosis with higher total energy.11 Resuscitation in the present case involved only a single shock at 300 joules.

Myocardial ischemia or necrosis secondary to the alternating current shock was probably the cause of the myocardial depression in this patient. The patient’s ventricular fibrillation and the finding of burns on both arms and the back confirm that the heart was on the current path. While the amount of current reaching the heart cannot be estimated in this case because current and length of exposure are unknown, the total energy delivered was probably substantial. In an experimental model, the likelihood of morphologic damage increased with alternating current discharge compared with direct current.12 Additionally, Lown et al13 have shown in a comparison of DC and AC defibrillation, that AC defibrillation is also much more likely to produce electrocardiographic changes consistent with myocardial ischemia. A similar mechanism may well have been operative in the present case. Regardless of mechanism, however, this case demonstrates that pulmonary edema resulting from electrical injury has a cardiac etiology. A noncardiogenic cause has yet to be substantiated.

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Right-to-Left Shunting Through a Patent Foramen Ovale Without Pulmonary Hypertension

Transient Improvement After Balloon Catheter Closure

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A patient had a history of right tuberculosis and severe hypoxemia secondary to right-to-left shunting through a patent foramen ovale without pulmonary hypertension. A balloon tip catheter was positioned in the left atrium and retracted against the atrial septum and the hypoxemia was temporarily resolved. (Chest 1990; 97:1250-52)

An occult probe patent foramen ovale is observed in up to 20 percent of the normal adult population without any significant clinical or hemodynamic complication. Acquired right-to-left shunting through a patent foramen ovale without pulmonary hypertension is quite a rare clinical phenomenon. This situation has already been reported, especially following pulmonary resection.14 We observed a case of severe hypoxia secondary to patent foramen ovale with normal pulmonary arterial pressures in a patient with a remote history of right pulmonary tuberculosis treated by collapse therapy.

CASE REPORT

A 67-year-old man was admitted to the hospital for evaluation of rapidly increasing dyspnea and severe fatigue. For two months prior to hospital admission he had noted a gradual onset of dyspnea with subjective improvement in the left lateral supine position.

Medical history of this coal worker included right pulmonary tuberculosis treated by collapse therapy in 1950, reticuloendotheliosis of the caviun treated by radiotherapy in 1966, and transient right hemiplegia in 1986. Physical examination on admission revealed an asthenic man with severe dyspnea at rest, perioral and finger cyanosis, and marked clubbing of finger and toes. Results of chest examination were normal. The first and second heart sounds were normal and there was no gallop rhythm. The blood pressure was 140/90 mm Hg. No sign of right ventricular failure was disclosed. Chest x-ray film showed posttuberculous right-sided retraction due to pleural symphysis and a slight mediastinal shift to the right (Fig 1). An electrocardiogram was within normal limits. The hemoglobin level was 16 mg/dl.

Arterial blood gas levels and an indwelling Swan-Ganz catheter results are summarized in Table 1.

Superior vena cava angiography disclosed no abnormal systemic venous return. Cardiac catheterization prior to pulmonary angiography disclosed a patent foramen ovale. Blood samples obtained in right and left pulmonary veins revealed normal PaO2 and oxygen saturation values except in the right inferior pulmonary vein because its catheterization was a failure.

The diagnosis of right-to-left shunt at atrial level was made conclusively by a right atrial angiogram, obtained by injection in

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Right-to-Left Shunting Through Patent Foramen Ovale (Remy-Jardin, Remy, Wallaert)