mm Hg (FiO₂, 0.5). Within 3 hs, no further deterioration occurred. On the same settings, the arterial blood gas levels showed the following; pH, 7.28; Pco₂, 42 mm Hg; Po₂, 60 mm Hg (FiO₂, 0.5). The patient was maintained on a single ventilator with a bifurcated endotracheal tube and the variable-resistance valve in place for the next 3 days, during which time the air leak via the BPF slowly decreased and ultimately resolved. He was then reintubated with a standard single-lumen endotracheal tube and maintained on the ventilator until he clinically improved.

**DISCUSSION**

The management of severe unilateral lung disease often is difficult because of the different physical characteristics of the two lungs. In these cases, the majority of the ventilator-delivered tidal volume enters the lung with the higher compliance and, as a result, insufficiently ventilates the contralateral lung, thus leading to worsening gas exchange or barotrauma. Patients with a large bronchopleural fistula may have the majority of the tidal volume delivered to the diseased lung and, subsequently, may lose a significant amount of volume via the airflow leak. In this situation, the nondiseased lung is insufficiently ventilated, and oxygenation and ventilation may be difficult to maintain. When the mechanical properties of the two lungs are different, synchronized or asynchronous ILV with two ventilators, or one ventilator with 2 separate breathing circuits, is occasionally attempted. Differential lung ventilation with two ventilators requires more extensive monitoring and is more labor-intensive. Concerns have been raised regarding impairments in venous return and oxygenation with the use of asynchronous dual ventilation in unilateral lung disease. Although synchronous ILV was not found to have an advantage over asynchronous ventilation in a dog model of unilateral acid-induced lung injury, it is unclear whether ventilator synchrony is important in the management of BPF.

We have designed and constructed a variable-resistance valve which has been tested in an animal model of BPF to demonstrate its efficacy in allowing differential lung ventilation, in a synchronized fashion, with the use of one ventilator. With a double-lumen endotracheal tube, the valve provides variable resistance to the lumen of the tube to one lung and, thus, controls the volume and airway pressure to that lung. We found that it effectively diverts a certain percentage of the ventilator-delivered tidal volume to the lung which is predominantly responsible for gas exchange, decreases the airway pressures to the injured lung, and, hence, decreases the air leak through an experimental BPF. The technique is comparatively simple and requires a less complex breathing circuit, as it allows for modification of tidal volumes, pressures, and flow rates by regulating a single valve to an optimal point between 0 and 100% occlusion. Other advantages of this method include a reduction in cost and improved ease of ventilator management because only one ventilator is used instead of two.

In this patient, BPF was successfully managed with differential lung ventilation using a variable-resistance valve in conjunction with a double-lumen endotracheal tube and a single ventilator. With the valve in place, the differential tidal volumes, flows, and pressures in each lung nearly matched the same parameters that were obtained when two ventilators were used. Therefore, the use of a variable-resistance valve and a single ventilator can be considered an alternative technique for the management of BPF. Further studies are needed in order to determine the efficacy of this technique in other forms of unilateral lung disease which require ILV.

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**Nonsustained Polymorphous Ventricular Tachycardia During Amiodarone Therapy for Atrial Fibrillation Complicating Cardiomyopathy**

**Management With Intravenous Magnesium Sulfate**

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A case is presented in which amiodarone was administered to suppress paroxysmal atrial fibrillation in a

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patient with an idiopathic cardiomyopathy. Eleven days after initiation of therapy with amiodarone, the patient experienced syncope and was noted to have recurrent episodes of polymorphous ventricular tachycardia. The patient was hospitalized and treated with a bolus as well as continuous infusion of intravenous magnesium sulfate. When the infusion was transiently discontinued, recurrences of polymorphous ventricular tachycardia were noted. The probable proarrhythmic action of amiodarone, although rare, is reviewed along with a discussion of the novel use of intravenous magnesium sulfate therapy. *(CHEST 1997; 111:1454-57)*

Although recognized with quinidine and other class Ia antiarrhythmic drugs, arrhythmia-aggravating potentials of amiodarone may be underrecognized. A case is presented in which polymorphous ventricular tachycardia developed during early administration of amiodarone to a patient for treatment of atrial fibrillation. The pathogenesis of this possible proarrhythmic action and its treatment with intravenous magnesium sulfate are discussed.

**Case Report**

A 77-year-old man with idiopathic cardiomyopathy and a left ventricular ejection fraction of 40% was hospitalized for management of heart failure and atrial fibrillation. Following an unsuccessful trial of procainamide, DC cardioversion was performed and sinus rhythm was restored. Results of thyroid function studies were within normal limits. Amiodarone therapy, 200 mg tid, was started and the patient was discharged on a regimen of captopril, hydralazine hydrochloride, warfarin sodium, furosemide, and potassium supplementation. Following cardioversion, the ECG revealed a left axis deviation, voltage consistent with left ventricular hypertrophy, and nonspecific repolarization changes. The QT interval was 390 ms, uncorrected, and 400 ms, corrected (Fig 1). After 11 days of therapy with amiodarone, the patient was hospitalized following a syncopal episode. Self-limited episodes of a polymorphous wide complex tachycardia were noted (Fig 2). Sinus bradycardia (55 beats per minute), biphasic T waves, and prominent U waves were present (Fig 3). The QTU interval had lengthened to 680 ms, uncorrected, and 600 ms, corrected. Hepatic enzymes, as well as serum potassium, magnesium, calcium, and creatinine levels were within normal ranges. Other than amiodarone, the patient was taking no drugs which increase the QT interval. The amiodarone level was 0.6 mg/L and was not elevated.

Polymorphous ventricular tachycardia, lasting up to 15 s in duration, subsequently was documented. Two grams of magnesium sulfate were given, and an infusion (4 g/250 mL D5W) was started at a rate of 90 mg (5 mL) per minute. Amiodarone therapy was discontinued, and no further episodes of tachycardia were observed. Within 24 h, the magnesium sulfate infusion was reduced to one quarter the strength. Attempts to discontinue the infusion over the next 48 h resulted in prompt recurrences of the same dysrhythmia; thus, the magnesium sulfate infusion was continued for 4 days. Following an additional 5 days, during which no additional recurrences were noted, the patient was discharged and antiarrhythmic therapy was discontinued.

**Discussion**

Despite uncontrolled trials which have reported substantial efficacy and safety with relatively low doses of

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**Figure 1.** ECG after restoration of sinus rhythm with DC shock therapy and prior to initiation of therapy with amiodarone. The recordings are "median complexes." From left to right, individual QRS complexes represent leads I, aVR, V1, V6 on row 1; leads II, aVL, V4, V5, V6 on row 2; and leads III, aVF, V3, V4 on row 3. The rhythm tracings on the righthand side are leads V1, II, and V6 from top to bottom. The paper speed associated with the rhythm recordings is 12.5 mm/s. The QT and QT corrected, intervals are 420 and 447 ms, respectively.

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amiodarone when used to suppress atrial fibrillation, this report highlights the agent’s potential proarrhythmic risk. Although the incidence of suspected proarrhythmia in a placebo-controlled trial of amiodarone has been negligible, an overall incidence of 1 to 7% has been reported.1

The occurrence of systemic and organ toxicities as well as of ventricular proarrhythmia has been reported to be higher in patients with poor ventricular function exposed to class Ia antiarrhythmic drugs. Potentiating factors from proarrhythmic events include bradycardias, electrolyte imbalances, and underlying disturbances of repolarization. Although of greatest concern when conventional agents are used, they similarly may occur with use of amiodarone and other class III antiarrhythmic agents.3

Compared with other antiarrhythmic agents, amiodarone has less of a negative inotropic effect. Amiodarone has beta and sodium channel blocking effects as well as class III properties due to inhibition of outward potassium currents during myocardial repolarization.4 In view of similar complex cellular actions to drugs with potential proarrhythmic actions, similar phenomena with amiodarone may be expected.

The nonspecific repolarization changes in this patient were not due to electrolyte imbalances and most probably reflected the underlying impaired myocardium. Amiodarone may have led to the development of early afterdepolarizations and abnormal-triggered activity, resulting in polymorphous ventricular tachycardia.

The invoked offending agent, amiodarone, was discontinued; however, episodes of polymorphous ventricular tachycardia continued for days when the infusion of magnesium sulfate was discontinued. Neither temporary pacing at a faster rate nor administration of intravenous isoproterenol were attempted. Although never before reported in such a situation, a bolus and a continuous infusion of intravenous magnesium sulfate were used to suppress episodes of polymorphous ventricular tachycardia. Intermittent discontinuation of the magnesium infusion was associated with recurrences of the polymorphous ventricular tachycardia; thus, spontaneous resolution of the dysrhythmia is unlikely to have occurred during this time period.

Several reports have supported the use of large boluses of intravenous magnesium to terminate and prevent recurrences of polymorphous ventricular tachycardia and torsades de pointes.5 The beneficial action of intravenous magnesium may be related to inhibition of

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21747/)

**Figure 2.** ECG at time of hospitalization following syncope after initiation of therapy with amiodarone.

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21747/)

**Figure 3.** Rhythm tracings recorded on telemetry after hospitalization. Short-long coupling intervals are followed by polymorphous ventricular tachycardia. This tracing was obtained after 11 days of loading with amiodarone. The total loading dose up to this point in time was 7,200 mg. The QTU and QTU, corrected, intervals were 650 and 600 ms, respectively.
early afterdepolarizations. In cellular preparations, as well as with monophasic action potential recordings, magnesium administration has been reported to suppress such activity. 

In conclusion, proarrhythmic actions may occur with use of amiodarone. Should polymorphous ventricular tachycardia develop in association with its use, the drug should be discontinued, electrolyte imbalances should be corrected, and pacing should be considered if necessary. Concomitantly, an infusion of intravenous magnesium sulfate should be administered until an adequate period of time has passed.

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Recurrent Alveolar Proteinosis Following Double Lung Transplantation*

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We present a case of recurrent alveolar proteinosis following double lung transplantation.

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Key words: alveolar proteinosis; lung transplantation

Abbreviations: PAP=pulmonary alveolar proteinosis

Pulmonary alveolar proteinosis (PAP) is a disorder characterized by excessive accumulation of surfactant-like material within the alveoli. Traditional management has been with bronchopulmonary lavage as needed for

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symptomatic relief. Progression to pulmonary fibrosis is rare.1 We present a case of a patient with PAP who progressed to pulmonary fibrosis (end-stage lung disease) treated with double lung transplantation who developed recurrent PAP in the transplanted lungs within 3 years of transplantation.

CASE REPORT

A 41-year-old woman was evaluated for lung transplantation because of end-stage lung secondary to PAP (Fig 1). Her disease had been diagnosed at age 27 years, and she had been treated with bronchopulmonary lavage (12 episodes) with initial good results but had a slowly declining course. Then, 8 years after diagnosis, she was referred to a major transplantation center for possible heart-lung transplantation. However, she experienced a spontaneous regression of symptoms and was not evaluated further. Approximately 6 years later, she was referred to our institution with worsening symptoms. On presentation, her FVC was 30% of predicted, her FEV1 was 35% of predicted, and her arterial blood oxygen saturation was 81% while breathing room air. During the ensuing 7 months, her condition steadily deteriorated and she underwent double lung transplantation. Postoperatively she did well. Routine follow-up was with chest radiography, pulmonary function tests, and surveillance bronchoscopic evaluation with transbronchial biopsy. Over the next 2 years, she experienced periodic episodes of bronchitis and developed mild obliterative bronchiolitis with concomitant decrease in pulmonary performance.

A little over 3 years after transplantation, she had a transbronchial biopsy of the right upper lobe to evaluate a new infiltrate seen on the chest radiograph (Fig 2). Specimens were negative for rejection or infection with fungi or a mycobacterial species. She returned in 2 months complaining of increasing fatigue and dyspnea on exertion. Another BAL and transbronchial biopsy were performed.

Histologic sections from the transbronchial lung biopsy re-