minished QRS amplitude, 3) decreased R-wave progression, and 4) inversion of precordial T waves.[8] The most important feature of ECG changes associated with a pneumothorax is the acute reversal of these changes after insertion of a chest tube.

We report this case as an example of tension pneumothorax mimicking myocardial ischemia to alert readers to include pneumothorax as part of the differential diagnosis of intraoperative ECG changes.

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Postoperative Unilateral Pulmonary Edema: Possible Amiodarone Pulmonary Toxicity

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Amiodarone is a highly effective antiarrhythmic drug for treating otherwise refractory ventricular and atrial arrhythmias. It has been shown to have numerous serious adverse side effects, including pulmonary toxicity.[1] The overall incidence of amiodarone pulmonary toxicity (APT) is estimated at 5–15% with a reported mortality rate of 5–10%.[1–4] The cause of APT is not clear. One theory is that amiodarone enhances free oxygen radical production in the lung, and that the free oxygen radicals in turn oxidize cellular proteins, membrane lipids, and nucleic acids. It is suggested that high fractions of inspired oxygen (FI_{O_2}) may accelerate these reactions.[5]

Some patients receiving amiodarone will eventually come to surgery for automatic internal cardiac defibrillator (AICD) insertion because of continued or recurrent refractory arrhythmias. This surgery is often performed *via* a left anterolateral thoracotomy requiring a period of one-lung ventilation. We report two cases of patients receiving amiodarone therapy who presented for this operation. The lungs of both patients were exposed to high FI_{O_2}s during one-lung ventilation, and on the continuously ventilated side both patients developed unilateral pulmonary infiltrates that, though not indisputably proven, were consistent with APT.

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Case Reports

Case 1. A 59-yr-old, 75-kg man with a long history of coronary artery disease, three previous myocardial infarctions, and two separate coronary bypass procedures presented for placement of an AICD because of multiple episodes of ventricular tachycardia.

Two years prior to admission, treatment with amiodarone 600 mg per day was begun. Despite this, during the 4 months prior to this admission he developed further episodes of ventricular tachycardia. The amiodarone was discontinued on the day of admission to the hos-

pital, and electrophysiologic testing revealed stable monomorphic ventricular tachycardia induced with a single β stimulus with a 465-ms cycle but accelerated to a second morphology with a 370-ms cycle on attempts to terminate the original ventricular tachycardia. Cardiac catheterization revealed a left ventricular ejection fraction of 15%. Right-side heart pressures and pulmonary vascular resistance were within normal limits.

An AICD was inserted 2 days later. General anesthesia consisted of thiopental 325 mg, pancuronium bromide 8 mg, sufentanil 0.1 mg, isoflurane 0.5–1.5%, and ventilation with an air–oxygen mixture and an initial FI_{O_2} of 0.6. Hemodynamic monitoring was performed with a brachial artery catheter and pulmonary artery and central venous catheters. The lungs were ventilated *via* a 37-Fr right double-lumen endobronchial tube. The patient was placed in a semisupine right lateral position. A left lateral thoracotomy was performed and the left lung deflated. The right lung was then ventilated with 100% oxygen. A large patch was placed on the posterolateral aspect of the left ventricle, and a small patch was placed high on the inferior right ventricle. The patches and leads were connected to a Ventak 15/50 AICD generator and the device was tested successfully. The left thoracotomy was closed, the left lung reexpanded, and the FI_{O_2} reduced to 0.6. The muscle relaxant was antagonized with glycopyrrolate 0.4 mg and pyridostigmine 20 mg, and the trachea was extubated in the operating room.

Two days postoperatively, the patient became febrile to 38.9° C, mildly confused, and hypoxic. Arterial blood gas analysis showed a Pa_{O_2} of 41 mmHg, a Pa_{CO_2} of 33 mmHg, and a hemoglobin oxygen saturation of 86% while the patient was breathing room air supplemented with oxygen 5 l/min *via* nasal cannula. Chest x-ray revealed an infiltrate in the mid-right lung field. The trachea was reintubated, and the lungs were mechanically ventilated. Prior to reintubation, there were no witnessed episodes of aspiration. White blood cell count at this time was 11,000 cells $\cdot$ cm^{-3}. Sputum and blood cultures taken at this time proved to be negative. In light of the patient's fever and pulmonary infiltrate, broad spectrum antibiotic coverage was instituted. Mechanical ventilation was instituted with an FI_{O_2} of 0.6 and a PEEP of 5 cmH_2O. A pulmonary artery catheter was reinserted with initial pulmonary artery pressures of 27/8 mmHg, a pulmonary artery wedge pressure of 6 mmHg, and a cardiac output of 5.9 l/min.

Four days postoperatively the entire right lung showed a diffuse infiltrate while the left lung remained relatively clear (fig. 1). For 3 days the right lung remained densely opacified. At bronchoscopy, a sterile brush and lavage were gram-stain negative for organisms and showed few white blood cells. Bacterial cultures were negative. Bronchoalveolar lavage cytology revealed foamy histiocytes compatible with amiodarone use. Amiodarone was discontinued and intravenous methylprednisolone, 100 mg four times per day, was begun with the presumptive diagnosis of APT. Gradually during the next 7–10 days the right lung infiltrate cleared. The trachea was extubated on postoperative day 17, and the patient recovered and was discharged in stable condition. Amiodarone was not restarted prior to discharge.

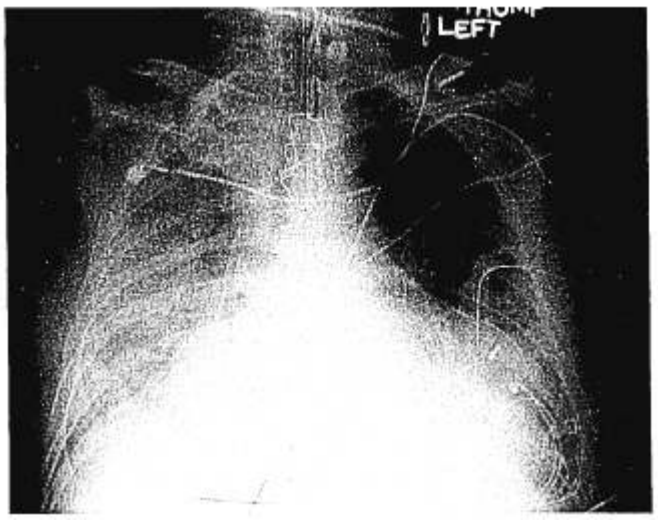

FIG. 1. Case 1, 4 days postoperatively.

Case 2. A 67-yr-old, 62-kg man with a history of coronary artery disease and recurrent ventricular tachycardia resistant to amiodarone therapy had an AICD placed without problems 1 month prior to this admission. The initial AICD placement was performed *via* a left anterior thoracotomy during anesthesia consisting of fentanyl 2.5 mg, diazepam 10 mg, and pancuronium 9 mg and an air–oxygen mixture. A 37-Fr left double-lumen endobronchial tube was used and one-lung ventilation was performed for approximately 1.5 h. Inspired oxygen concentration was maintained at less than 50% during the entire operation, and hemoglobin oxygen saturations as low as 89% were tolerated during one-lung ventilation.

The AICD initially worked well, but the posterior cardiac patch became displaced and the patient developed additional episodes of ventricular tachycardia requiring repeated cardioversions. He was readmitted for repositioning of the AICD patch. He was still taking amiodarone 200 mg alternating with 300 mg daily. His lung fields on physical examination and chest x-ray were noted to be clear.

The patient was taken to the operating room for a planned left thoracotomy and replacement of the AICD patch. Anesthesia consisted of diazepam 20 mg, pancuronium 10 mg, fentanyl 2.0 mg, and nitrous oxide and oxygen in a 50:50 mixture with isoflurane up to 1.5%. The lungs were ventilated *via* a 37-Fr right double-lumen endobronchial tube. Hemodynamic monitoring was performed with radial arterial, central venous, and pulmonary artery catheters.

During placement of the AICD patch (a period of 2 h), the left lung was deflated and the right lung was ventilated with 100% oxygen. The operation proceeded uneventfully, and at the end of the case the double-lumen endotracheal tube was replaced with a 7.5-mm cuffed tube and the patient's lungs electively ventilated with an FI_{O_2} of 0.5 and a PEEP of 3 cmH_2O. The first postoperative chest x-ray showed atelectasis of the left lower lobe; the right lung field was clear. The next day the patient improved, and the trachea was extubated without incident. He was administered 40% oxygen *via* face mask, which was subsequently reduced to 2 l/min by nasal cannula.

Three days postoperatively the patient developed hypoxia, dyspnea, and fever to 38.3° C, and the chest x-ray revealed an extensive infiltrate in the right lung (fig. 2). Increasing the patient's oxygen supplementation to 4 l/min by nasal cannula relieved the dyspnea and maintained oxyhemoglobin saturation by pulse oximetry above 90%. No episodes of gastric aspiration had been witnessed. No bacteria could be cultured from sputum. Blood cultures were negative. White blood cell count was 7,300 cells $\cdot$ cm^{-3}. No bronchoscopy or lavage was performed in this patient. The diagnosis of APT was suggested, and a 7-day course of prednisone was begun. Amiodarone was continued at 200 mg/day. The right lung infiltrate slowly resolved during the next 10 days, and the patient was discharged in good condition.

DISCUSSION

There appears to be two distinct types of presentation of patients with APT.[1] The more common one consists of a slow insidious onset of progressive dyspnea, cough, weight loss, and infiltrates on chest x-ray. The second type, which applies to our two cases presented here, has

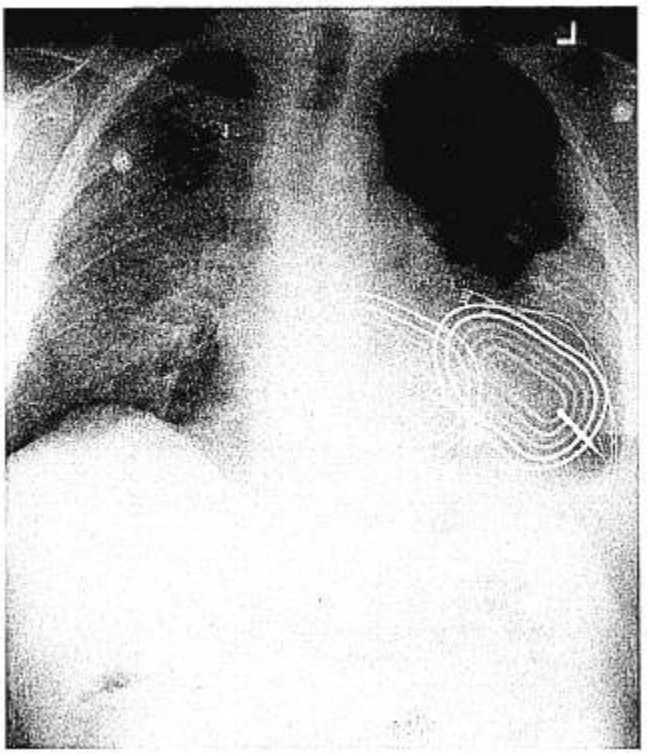

FIG. 2. Case 2, 3 days postoperatively.

a much more acute onset of dyspnea, cough, hypoxemia, and occasionally fever that may even mimic an infectious pneumonia. The acuteness of onset may suggest the diagnosis of a pulmonary embolus or congestive heart failure, and, in fact, some authors advocate aggressive diagnostic interventions to rule out these and other possible diagnoses in this patient population.[6]

APT remains a clinical diagnosis of exclusion. Regardless of the type of presentation, certain signs and symptoms are common. These include: 1) dyspnea and dry cough; 2) rales on physical examination; 3) some degree of hypoxemia on arterial blood gas analysis; 4) diffuse pulmonary infiltrates or infiltrates mainly in the upper lung fields on chest x-ray; and 5) improvement after stopping or reducing the dose of amiodarone with or without steroid therapy.[1-3] Fever, malaise, rales, pleural effusions and pleural rubs are much less common, but all have been reported. Biopsy evidence of alveolitis and bronchoalveolar lavage revealing characteristic "foamy histiocytes," as seen in case 1, are considered adjuncts to diagnosis as well.[1,7] These foamy histiocytes may be found in both acute and chronic types of APT and are believed to be either alveolar macrophages or transformed type II pneumonocytes laden with lamellated phospholipid inclusion bodies. While they may be preset in patients without symptoms of APT, they are a reproducible feature of lung injury associated with amiodarone.[8] Currently, however, there are no specific clinical, radiographic, or pathologic findings considered pathognomonic for the condition.

Predicting which patients will develop APT has been difficult.[3] Presently, there are insufficient studies to describe a distinctly different set of risk factors for the development of acute APT *versus* chronic APT. There does appear to be a correlation between the daily and cumulative total dose and the appearance of APT. Several authors have suggested that patients receiving daily amiodarone doses of less than 400 mg or cumulative doses of less than 100 g are at reduced risk of developing APT compared to those patients with higher daily and total doses.[9,10] Still, there are multiple reports of apparent APT in patients receiving smaller doses.[11] Other proposed predictors of developing pulmonary toxicity include an abnormal preadministration chest x-ray, abnormal pulmonary function tests, decreased carbon monoxide diffusing capacity, and a wider than expected alveolar–arterial oxygen gradient on arterial blood gas analysis. One study of nonsurgical patients found a 10-fold greater incidence of chronic-type APT in patients with both an abnormal pretreatment chest x-ray and low carbon monoxide diffusing capacity.[12] Another study of 56 patients who underwent cardiac operations found that patients who had received amiodarone therapy preoperatively were more likely to experience postoperative respiratory failure than were those in whom amiodarone was first administered immediately postoperatively.[13] In addition, patients who were receiving amiodarone preoperatively and developed postoperative respiratory failure had significantly increased alveolar–arterial oxygen gradients compared to the amiodarone treated patients who did not develop respiratory failure.[13]

The mechanisms of APT have been studied, but at this time there is still no clear-cut consensus as to the actual cause. Mechanisms hypothesized include: 1) direct toxicity secondary to high drug concentrations in pulmonary tissues[14,15]; 2) inhibition of phospholipases and subsequent deposition of phospholipids in lung parenchyma[16]; 3) hypersensitivity reactions to the drug or metabolites[12,17]; and 4) drug enhancement of oxygen free radical production and secondary cellular injury.[16,18,19] The actual mechanism of lung injury is probably some combination of all of these.

While there have been a multitude of case reports of APT, only a few postoperative cases have been reported. In 1988, Kay *et al.*[5] reported four postoperative cases of presumed APT. This was part of a larger study of side effects of amiodarone use that included 288 patients receiving amiodarone, 33 of whom underwent surgery for various procedures. Only 1 of the 4 patients who developed APT underwent an intrathoracic procedure. The exact intraoperative FI_{O_2} used in each case was not men-

tioned. In all 4 patients the tracheas were extubated easily at the end of surgery or shortly thereafter, but dyspnea and hypoxia required reintubation 18–72 h postoperatively. Systemic and pulmonary arterial pressures as well as cardiac outputs were reported as normal. Bacterial and fungal cultures were negative. Two of the 4 patients eventually died secondary to their pulmonary complications. *Post mortem* examination of the lungs of these two patients revealed intraalveolar foamy macrophages, also noted in one of the two cases we have reported. The authors reasoned that the consistent temporal pattern of onset of pulmonary abnormalities suggested exposure to a common factor in the pathogenesis of the APT. They concluded that the common intraoperative factor was a high $F_{I_{O_2}}$.[5] The authors did not, however, show a significant difference between the intraoperative $F_{I_{O_2}}$s of the 4 patients who developed APT and the intraoperative $F_{I_{O_2}}$ of the other 29 patients without postoperative pulmonary problems.

In 1989, Kupferschmid *et al.*[9] described, retrospectively, 71 patients operated on for obstructive hypertrophic cardiomyopathy. Operations performed were either left ventricular myomectomy or mitral valve replacement in all patients. Neither the intraoperative or the postoperative $F_{I_{O_2}}$s used were reported. Sixteen of these patients were receiving amiodarone preoperatively. Four of these 16 patients suffered pulmonary dysfunction, described as moderate in 1 and as acute respiratory distress syndrome in the remaining 3, necessitating a 4-fold increase in the number of days of ventilator support. None of the patients not receiving amiodarone (n = 55) required more than 24 h of ventilator support. The authors recommended that patients for this surgery should have as long a drug-free interval as possible prior to surgery and that these patients be informed of the increased risk of postoperative complications, especially those patients having received more than 100 g of amiodarone or having been treated for greater than 300 days.

The two probable cases of unilateral APT we have presented here support previous concerns over a high $F_{I_{O_2}}$ in patients receiving amiodarone undergoing general anesthesia. Our review of the literature suggests that there may be some interventions pre- and intraoperatively that we can undertake to decrease the incidence of postoperative APT. Discontinuing amiodarone administration prior to surgery is desirable but may not be possible especially in patients such as ours, presenting for AICD placement. A different surgical approach to the heart may be suggested for AICD placement in order to avoid the need for one-lung ventilation. Most importantly, it would appear prudent to restrict the inspired oxygen concentration in patients receiving amiodarone undergoing general anesthesia to the lowest level capable of maintaining adequate systemic oxygenation.

Our future management of these patients will reflect our belief that this may be a real phenomenon. However, we wish the reader to realize that no animal model as yet has established a cause-and-effect relationship between oral amiodarone administration and secondary oxygen-enhanced pulmonary toxicity, and, thus, this association is scientifically circumstantial at present. Further investigation in an animal model is needed.

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Intrathecal Fentanyl Alleviates Spasticity in the Presence of Tolerance to Intrathecal Baclofen

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Injury to the central nervous system can result in spasticity in the extremities distal to the site of neurologic damage. Spasticity usually requires treatment because it interferes with patients' ability to position satisfactorily, causes discomfort, and hampers patient transfers. Spasticity is usually adequately controlled with oral medications such as baclofen[1] or benzodiazepines.[2] When the spasticity is refractory to oral agents, it may be treated with an implanted drug delivery system that continuously infuses baclofen into the cerebrospinal fluid.[3] Continuous infusion of morphine into the cerebrospinal fluid has also been used to treat spasticity.[4] Tolerance may develop to both baclofen and morphine administered intrathecally, and an increasing amount of drug may be required to provide satisfactory control of spasticity.[5,6] This report describes the successful relief of spasticity with intrathecal fentanyl in a patient whose spasticity was resistant to chronically administered intrathecal baclofen.

CASE REPORT

A 42-yr-old man with a 23-yr history of an incomplete spinal cord injury at the fifth cervical level was referred to the Pain Service for evaluation of spasticity in May 1989. His spasticity had been effectively managed with oral baclofen until 5 yr previously, when increasing spasticity required treatment with a combination of oral baclofen, diazepam, and clonidine. At the time of referral he was refractory to these medications. Neurologic evaluation and magnetic resonance imaging failed to disclose pathology that would account for the increase in his symptoms. The patient's spasticity was interfering with wheelchair transfer, preventing him from sitting upright, and contributing to the development of decubitus ulceration.

Consequently the patient was referred for evaluation and treatment of his spasticity. The patient received intrathecal baclofen 50 μg and fentanyl 40 μg administered 1 week apart. The response to each agent was evaluated by clinical examination and with the flexion dynamometer, an instrument designed to measure and document spasticity accurately.[7] Baclofen and fentanyl both produced dramatic temporary reduction in spasticity (fig. 1). In August 1989 a spinal pump (Medtronics Inc.) was subcutaneously implanted. The pump infuses baclofen intrathecally at a continuous rate. The infusion rate can be adjusted electrically using a programing computer and radiofrequency interface similar to that used to encode a cardiac pacemaker. The patient's response to the intrathecal baclofen infusion at the rate of 200 μg/24 h was cessation of spasticity and profound lower extremity flaccidity. Subsequently the rate of delivery of baclofen was reduced to 140 μg/24 h to provide enough muscle tone to facilitate transfers.

The daily requirement of baclofen increased steadily over the past 12 months until the patient was currently receiving 720 μg of baclofen every 24 h. Despite the increased baclofen requirements the patient's spasticity persisted. The pump was examined according to the manufacturer's recommendations and was found to be in proper working order. In April 1991 the patient inadvertently received an overdose of intrathecal baclofen (1,440 μg/24 h) at twice the intended infusion rate. In the ensuing 12 h the patient subsequently noted the progressive absence of spasticity, weakness in his arms, and difficulty breathing. He observed progressive weakness affecting his legs initially, and then his arms, and finally his muscles of respiration. The pump was turned off, and the patient was admitted to the hospital for observation. He was alert and oriented. Pulmonary function tests were performed and the patient underwent clinical examination and evaluation with the flexion dynamometer, which confirmed physical examination findings of profound lower limb flaccidity. His symptoms improved over the next 4 h; the pump was reprogrammed to deliver the intended 24-h dose of baclofen, 700 μg; and the patient was discharged from the hospital.

Within 24 h he noted a return of spasticity, and he returned 1 week later for an increase in the baclofen infusion rate. Prior to adjusting the pump, fentanyl 40 μg was injected into the cerebrospinal fluid, causing a marked diminution in spasticity within 30 min. Figure 1 is a graphic representation of the flexion dynamometer spasticity measurements performed over the preceding 18-month period including the initial evaluation.

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