cardia, and electromechanical dissociation are useful features in the clinical diagnosis of this condition. Our patient was asymptomatic, and no signs of tamponade were present at the time a moderate pericardial effusion was demonstrable by echocardiography. Because the patient's clinical state was stable, conservative management was instituted. Careful serial clinical examination until the time of death failed to reveal evidence of tamponade.

A possible explanation for the sequence of events observed in our patient is myocardial rupture with consequent hemopericardium and partial sealing off of the site of the tear. The observations of Lautsch and Lanks, who found organizing thrombus at the site of rupture in 65 percent of 43 cases studied, support our thesis. A slow leak of blood may have occurred over the next few days, and a second large hemorrhage resulted in cardiac tamponade. Based on this experience, we recommend pericardiocentesis in patients who develop moderate or large pericardial effusions following acute myocardial infarction. If hemopericardium is present and no precipitating cause (eg, anticoagulant therapy) is evident, myocardial rupture should be considered and appropriate surgical therapy instituted. Cardiac rupture should be especially considered if pericardial effusion occurs in the setting of new or protracted chest pain and electromechanical dissociation, preceded or accompanied by S-T segment elevation or depression.

Successful surgical treatment of myocardial rupture was reported by Cobbs and associates. Their patients presented with acute cardiac tamponade. Two of their patients who had surgical treatment have been followed-up for one and three years respectively.

Rupture of the heart can present as an asymptomatic pericardial effusion. Because of the instability of the situation, expeditious surgical correction of the rupture is recommended.

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**Adult Respiratory Distress Syndrome following Administration of Lidocaine**

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A patient twice developed the adult respiratory distress syndrome following an adverse reaction to administration of lidocaine. To our knowledge, such an association has not been reported previously.

Adverse reactions to therapy with lidocaine occur infrequently and are usually related to the inadvertent administration of toxic doses of the drug. Occurrence of the adult respiratory distress syndrome (ARDS) associated with such a reaction has not been described. The present report is that of a woman who twice developed ARDS following administration of lidocaine.

**CASE REPORT**

A 57-year-old woman was to undergo fiberoptic bronchoscopy and transbronchial biopsy for the evaluation of a right upper lobe mass. Her past history included a left mastectomy for carcinoma. She had no history of cardiac, renal or hepatic dysfunction. She denied any history of drug allergies.

She was premedicated with meperidine 50 mg and atropine 0.4 mg intramuscularly 30 minutes prior to the bronchoscopic procedure. Anesthesia of the oropharynx was begun using 5 ml of 4 percent lidocaine by ultrasonic nebulization, to be administered over seven minutes. Five minutes after nebulization was initiated, however, the patient became somnolent and had several generalized tonic-clonic seizures which abated after the administration of diazepam 5 mg intravenously. She was then noted to have labored respirations, audible wheezing, and was cyanotic. There was no evidence of vomiting or aspiration. She was intubated orally without difficulty. A large quantity of pink, frothy secretions was suctioned from the endotracheal tube. Upon arrival at the respiratory ICU, she was afebrile with a blood pressure of 80/60 mm Hg, a pulse rate of 120 beats per minute and regular. There was no jugular venous distention; diffuse inspiratory and expiratory rales were heard throughout all lung fields. The chest film revealed diffuse interstitial and alveolar infiltrates (Fig 1). Arterial blood gas levels on a fractional inspired oxygen concentration (Fio2) of 1.0 and 5 cm of positive end-expiratory pressure (PEEP) were as follows: PaO2 66 mm Hg; PaCO2 37 mm Hg; pH 7.48. A balloon-tipped, flow-directed thermocatheter was placed and revealed a pulmonary capillary wedge pressure of 10 mm Hg. Static lung compliance was 0.02 liter per cm H2O. The patient improved rapidly, and arterial blood gas determinations 48 hours later, when she was alert and breathing spontaneously on a T-tube with a supplemental oxygen flow rate of 5 liters per minute, were as follows: PaO2 84 mm Hg; PaCO2 37 mm Hg; pH 7.42. Her chest film showed marked clearing of the infiltrates (Fig 2), and she was extubated without difficulty.

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relatively infrequent in the medical literature, and there is no known case of lidocaine hypersensitivity reaction in connection with fiberoptic bronchoscopy. There are a number of possible mechanisms that could explain these episodes in our patient. Diffuse alveolar filling process due to aspiration pneumonia could resemble the findings in our patient; however, there was no evidence of vomiting and the patient was afebrile throughout the course. Drug toxicity or drug idiosyncracy especially with the occurrence of seizures preceded by preconvulsive somnolence is a strong possibility. However, the second episode was not associated with seizures and there was no evidence of eosinophilia. Post-ictal pulmonary edema would explain the first episode, but could not explain the second. Other agents which are known to cause the adult respiratory distress syndrome, such as chlorothiazide, nitrofurantoin or propoxyphene, were not used in our patient. Early discontinuation of PEEP may have fortuitously allowed the primary process to recur; however, 24 hours after discontinuation of PEEP, the chest roentgenogram was clear. Anaphylactic reaction seems the only mechanism that could explain the clinical features of both episodes and the development of ARDS following each administration of lidocaine. The presence of normal pulmonary arterial wedge pressures was consistent with a noncardiogenic etiology.

It has been established that various chemical mediators are liberated during anaphylaxis. These include histamine and slow-reacting substance A, which are known to cause increased capillary membrane permeability. We believe that the development of ARDS in our patient was due to an anaphylactic reaction to the administration of lidocaine.

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Figure 1. Chest film reveals diffuse interstitial and alveolar infiltrates.

Three days after the first episode, 0.5 ml of 1 percent lidocaine was inadvertently given subcutaneously for local anesthesia prior to replacement of an arterial catheter. Five minutes later she became agitated, cyanotic and tachypneic with a blood pressure of 80/60 mm Hg. There was no evidence of aspiration or seizure activities. She was placed back on the ventilator and, when arterial blood gas levels were determined after the no, was increased to 1.0, the Po2 had dropped to 53 mm Hg. A repeat chest film showed new fluffy bilateral alveolar infiltrates. The pulmonary capillary wedge pressure was 9 mm Hg; static compliance was 0.017 liter per cm H2O. During the next 48 hours, oxygenation improved markedly, and the alveolar infiltrates almost totally cleared. She was extubated the following day and seven days later underwent fiberoptic bronchoscopy with cocaine anesthesia. A transbronchial biopsy showed that the right upper lobe mass was an undifferentiated adenocarcinoma.

DISCUSSION

Reported cases of hypersensitivity to lidocaine are

Figure 2. Marked clearing of the infiltrates is noted.

CHEST, 81: 5, MAY, 1982

ARDS FOLLOWING ADMINISTRATION OF LIDOCAINE 645