local anesthesia. The obvious concern of causing symptomatic intraabdominal tumor implantation did not develop in our patient and has not been reported elsewhere. Cimochowski and associates6 have reported successful use with malignant effusions for periods exceeding one year.

Malignant effusion should be considered when delayed contralateral mediastinal shift occurs postpneumonectomy and pleuroperitoneal shunting is an effective treatment alternative in symptomatic patients with this problem.

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Previously Unreported Adverse Reaction to Encainide*

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We describe a patient with recurrent atrial fibrillation who suffered a previously unreported adverse reaction to encainide therapy manifested by fever, chills, diaphoresis and myalgia. The patient had a similar response upon rechallenging with encainide, which resolved on discontinue of therapy. (Chest 1989; 96:838-89)

Several new antiarrhythmia agents have been approved for clinical use recently.1 An ideal antiarrhythmia agent should demonstrate a high degree of clinical efficacy with minimal side effects and a high therapeutic/toxicity ratio. Encainide (±)-4-methyl-N-[2-[2-(1-methyl-2-piperidinyl)ethyl]-phenyl] benzamide hydrochloride is a new class 1C antiarrhythmia agent.2-3 The absorption is rapid and complete from the gastrointestinal tract and it undergoes extensive hepatic degradation to form at least four metabolites. Two of these metabolites: O-demethylencainide and 3-methoxyl-0-demethylencainide have been shown to be more active and contribute to the antiarrhythmic effects.4-5 Encainide and its metabolites are excreted by the kidneys; elimination half-life varies extensively. It undergoes a polymorphic pattern of oxidation metabolism in the liver with two different phenotypes: (1) extensive metabolizers and (2) poor metabolizers. Extensive metabolizers comprise the majority of the population and 7 to 10 percent are poor metabolizers. Encainide half-life in extensive metabolizers is 2.34 to 2.47 h and in poor metabolizers is 8.7 to 11.28 h.6 Common adverse effects include proarrhythmic effects, dizziness, blurred and abnormal vision and headaches. To the best of our knowledge, fever, diaphoresis and myalgia have not been reported. Therefore, we are reporting a patient who developed fever, chills and intense myalgia within 2 h of receiving encainide therapy. Rechallenge with the drug produced the same reaction.

CASE REPORT

A 55-year-old white man presented in 1985 with palpitations secondary to atrial fibrillation and rapid ventricular response. Physical examination at that time was unremarkable except for a systolic ejection murmur along the left sternal border. An echocardiogram revealed normal left atrial and left ventricular internal dimensions with no evidence of left ventricular hypertrophy. The aortic valve was calcified with slightly decreased leaflet excursion. He was successfully electrically cardioverted to normal sinus rhythm and discharged on sustained release procainamide (Pronestyl), 750 mg every 6 h, and digoxin, 0.25 mg daily. Four months later, he developed arthralgia and synovitis consistent with lupus-like syndrome and procainamide was discontinued. He remained asymptomatic since then on digoxin 0.25 mg daily until late July 1987, when he developed progressively severe exertional dyspnea and diaphoresis. He was hospitalized in August 1987 and was found to be in atrial fibrillation with satisfactory ventricular response. Again, he underwent successful electrical cardioversion after a bolus of 500 mg of intravenous Pronestyl followed by a maintenance dose of 2 μg/min. Following discontinuation of intravenous Pronestyl, oral Pronestyl was not restarted in view of the history of lupus-like syndrome and subsequently he was started on encainide, 25 mg orally on August 12, 1987. One and a half hours later on the same day, he developed fever of 38.2°C associated with chills, diaphoresis and severe calf muscle discomfort and backache. There were no pruritus, skin rash, joint pains or abdominal complaints. Fever, chills and diaphoresis subsided in 2 h but he continued to have myalgia for 10 to 12 h. He received a second dose of encainide, 25 mg, at 8:30 AM on August 13, 1987, and at 10:30 AM, he again developed similar symptoms of fever (39.2°C), chills and diaphoresis which lasted for 2 h and severe myalgia in calf muscles and back that persisted for 10 to 12 h. Laboratory evaluation performed on the same day, approximately 10 h after the second dose of encainide, revealed a sedimentation rate of 5 mm/h. Serial creatinine phosphokinase values were 40 and 43 IU/L, with 100 percent MM fraction. Antinuclear antibody and rheumatoid factors were negative. Serum cryofibrinogen was positive and cryoglobulin was negative. Complement studies revealed C4 of 158 mg/dL (normal range, 70 to 176 mg/dL), C3 of 22 mg/dL (normal range, 14 to 45 mg/dL) and CH50 of 22/4/2 (normal range, 28 to 84/4). Encainide was discontinued with no recurrence of symptoms and he was subsequently discharged in sinus rhythm on diltiazem and digoxin.

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Adverse Reaction to Encainide (Goli-Bijanki et al)
Discussion

This case illustrates a previously unreported side effect of encainide, an adverse reaction which occurred 1/2 h after the initiation of therapy. Because he had a similar response to encainide upon "rechallenging," we feel that this is most likely secondary to encainide therapy, especially since the symptoms resolved on discontinuing the therapy. The significance of borderline low CH50 is uncertain. The likelihood of an immune reaction involving the complement pathway seems remote since C3 and C4 were in the normal range. However, serial measurements of complement and tests for immune complexes were not done. On review of the literature,\textsuperscript{14} the reported side effects with encainide therapy are: congestive heart failure (1 percent), abnormal and blurred vision (10 percent), dizziness (7 percent), proarrhythmia (10 percent), ventricular tachycardia (6 percent), QRS prolongation, (9 percent), atrioventricular block (3 percent), headache, nausea, and taste perversion (2 percent), asthenia, ataxia, dyspnea, palpitations, paresthesia, vertigo, vomiting (1 percent) and rare reports of abnormal liver function tests, hepatitis, jaundice and death.

References


Respiratory Muscle Fatigue from Functional Upper Airway Obstruction\textsuperscript{a}

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A case of functional upper airway obstruction is presented. The case is unusual because even though no identifiable organic cause could be found for dyspnea and stridor, the patient developed respiratory failure from respiratory muscle fatigue.

(Chest 1989; 96:689-90)

In recent years, there have been several reports of patients with nonorganic laryngeal dysfunction presenting as asthma\textsuperscript{1,2} or upper airway obstruction.\textsuperscript{3,4} In some cases, respiratory distress is severe enough to require endotracheal intubation\textsuperscript{5} or tracheostomy.\textsuperscript{6} Arterial blood gas levels are usually normal\textsuperscript{7} or show a respiratory alkalosis with a normal alveolar-arterial oxygen difference.\textsuperscript{1,2} Severe hypoxemia without hypoventilation or respiratory acidosis has also been reported.\textsuperscript{8} We describe a patient with severe respiratory distress without an apparent organic cause who developed clinical signs of respiratory muscle fatigue, followed by respiratory acidosis requiring intubation and mechanical ventilation. To our knowledge, respiratory failure from respiratory muscle fatigue has not previously been reported in a patient with functional upper airway obstruction.

Case Report

A 32-year-old woman was admitted with the chief complaint of shortness of breath for three days. Two months earlier, she had been admitted to another hospital with the diagnosis of asthma. She improved with treatment and was discharged on oral therapy with theophylline and an inhaled \beta\textsubscript{2}-adrenergic agonist. At the time of this admission, the patient had noted three weeks of dry cough and three days of worsening dyspnea. There was no history of fever or chills.

Physical examination revealed an obese woman in moderate distress with accessory muscle use and audible wheezes. Temperature was 36.3°C (97.3°F), blood pressure was 140/90 mm Hg, the heart rate was 140 beats per minute, and the respiratory rate was 50 breaths per minute. Expiratory wheezes were heard over the trachea, and the pulmonary fields were clear. Findings from the remainder of the examination were within normal limits. Laboratory findings disclosed the following values: white blood cell count, 14,300/cu mm, with a normal differential cell count; sodium, 139 mEq/L; chloride, 106 mEq/L; carbon dioxide, 15.9 mmol/L; potassium, 3.3 mEq/L; glucose, 177 mg/dl; and creatinine, 1.2 mg/dl. The anion gap was 17. Other values from blood chemistry were normal. An arterial blood gas analysis, performed when the patient was breathing room air, revealed a pH of 7.40, PaCO\textsubscript{2} of 21 mm Hg, and PaO\textsubscript{2} of 112 mm Hg. Gram stain of sputum revealed numerous polymorphonuclear leukocytes and mixed Gram-positive and Gram-negative organisms. The findings from urinalysis were normal. The chest x-ray film revealed diminished pulmonary volumes and was without infiltrate. An ECG showed a sinus

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