bilateral otorrhea. Otolaryngologic evaluation showed bilateral tympanic perforations, without a history of previous tympanic or ear problems.

DISCUSSION

CPAP is an effective and simple method to increase arterial oxygenation in nonintubated patients. In this case, intubation was required for hypercapnia, although arterial oxygenation improved with CPAP and increased FiO2.

There are many complications associated with CPAP, including subconjunctival emphysema, pulmonary venous and systemic gas embolism, and corneal abrasions. Pneumocephalus has been reported in association with CPAP in a patient with an unrecognized basilar skull fracture.

This case reports bilateral tympanic membrane rupture and otorrhea associated with CPAP. It is probable that the patient ruptured his tympanic membranes by coughing against the CPAP. Others have reported the need for low resistance expiratory circuits and have demonstrated that the Downs valve we used has low expiratory flow resistance. A trans tympanic pressure of 724 to 2,320 cmH2O is necessary for tympanic rupture with normal tympanic membranes. At no time was the CPAP this high unless a transient, high pressure impulse occurred that resulted in tympanic rupture but was not reflected on the manometer.

Bilateral tympanic membrane rupture and otorrhea can be added to the complications of CPAP in patients who are agitated and coughing. We recommend cautious use of CPAP in a coughing and agitated patient.

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REFERENCES


Rapid Suppression of Flecainide-Induced Incessant Ventricular Tachycardia with High-Dose Intravenous Amiodarone*

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A severe case of flecainide-induced incessant ventricular arrhythmias is presented. These arrhythmias were resistant to various intravenous antiarrhythmic drugs and to cardiac pacing. Intravenous amiodarone administered over a short period and in a high dose strikingly abolished all ventricular arrhythmias.

The proarrhythmic potential of flecainide acetate, a class Ic antiarrhythmic agent, has been well documented in the last few years. It is estimated to occur in 6.8 percent of the patients overall and was serious in 2.3 percent and lethal in 1 percent. We herein report the findings in a patient with flecainide-induced incessant symptomatic ventricular tachycardia, in whom we observed a striking and immediate response to high-dose intravenous amiodarone, with total abolition of ventricular tachycardia.

CASE REPORT

A 62-year-old man with a history of anterior myocardial infarction and paroxysmal sustained ventricular tachycardia was admitted following the documentation of symptomatic (dizziness and presyncope) nonsustained ventricular tachycardia. He was receiving amiodarone (200 mg/day) and mexiletine (600 mg/day). Physical examination revealed mild signs of heart failure. The electrocardiogram revealed multiform premature ventricular complexes. The P-R interval and QRS duration were normal, as were the Q-T and QTc intervals (0.36 second and 0.34 second, respectively). A two-dimensional echocardiogram showed a dilated and poorly contractile left ventricle.

In view of the ineffectiveness of the drugs, all prior antiarrhythmic therapy was withdrawn (two weeks). Therapeutic trials with quinidine and propafenone were unsuccessful, with persistence of complex premature ventricular contractions and nonsustained ventricular tachycardia. Oral flecainide (100 mg twice daily) was administered without improvement. The dosage was then gradually increased every four days up to 200 mg twice daily. An increase in the number of complex premature ventricular contractions and nonsustained ventricular tachycardia was documented by Holter monitoring. There was no significant prolongation of the P-R, QRS and Q-T intervals and no electrolyte disturbances were detected.

After 48 hours of therapy with flecainide (400 mg/day), the patient developed almost incessant self-sustained ventricular tachycardia with two distinct morphologies and rates (Fig 1). These two morphologies of ventricular tachycardia were different from the previously observed paroxysmal sustained and nonsustained ventricular tachycardia and were considered to be nonclinical. During a period of eight hours, the patient suffered recurrent episodes of sustained ventricular tachycardia despite therapy with intravenous lidocaine (100-mg bolus), bretylium tosylate (bolus of 5 mg/kg), and procaínamide hydrochloride (stopped after 200 mg because of hypotension). Faceting techniques failed to terminate the ventricular tachycardia. Because of hemodynamic deterioration, cardiocorversion became

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