Although the usefulness of amantadine in the treatment of transplant patients with established influenza A infections is clear, the prophylactic use of amantadine in high-risk transplant patients has not been evaluated, despite being theoretically appealing. In cardiac transplant patients who require OKT3 or anti-thymocyte globulin for treatment of rejection during influenza A epidemics, and in patients who are not vaccinated or do not exhibit adequate antibody titers following vaccination, the prophylactic administration of amantadine may reduce the incidence and severity of influenza A infections. A regimen of amantadine, 100 mg orally twice daily, during antilymphocyte therapy and for two weeks thereafter until the patient's T lymphocyte function recovers, would seem logical. Such prophylactic treatment cannot be firmly endorsed, however, until an appropriate randomized, prospective study has been completed.

References

Myocardial Stunning Following Respiratory Arrest*

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Myocardial stunning is defined as a prolonged myocardial dysfunction with gradual return of contractile activity after a brief episode of severe ischemia. Usually it is seen in patients with myocardial infarction following treatment with thrombolytic agents, in patients with angina, and in patients recovering from cardiopulmonary bypass surgery. We report an interesting case of myocardial stunning following respiratory arrest. (CHEST 1995; 108:1459-60)

Keywords: myocardium; respiratory arrest; resuscitation; stunning

Herein is the report of a case of myocardial stunning after respiratory arrest.

Case Report

Sudden respiratory arrest following intravenous injection of di-azeptam and methohexital developed in a 24-year-old previously healthy white woman during a dental procedure. She became cyanotic and was successfully intubated within 3 to 4 min. The cardiac monitoring at that time revealed a normal sinus rhythm, and she was transferred to Nassau County Medical Center. At the time of arrival in the emergency department, findings of the physical examination were as follows: pulse, 90 beats per minute; BP, 80/50 mm Hg; temperature, 35.5°C; and spontaneous respiratory rate, 20 breaths per minute. The remainder of the physical examination disclosed no abnormalities. At the time of admission, laboratory values, including complete and differential WBC counts and renal and hepatic profiles, were normal. The arterial blood gas level with an FiO2 of 1.0 revealed a pH value of 7.52, a PaO2 value of 554 mm Hg, and a PaCO2 level of 32 mm Hg. A 12-lead ECG was normal except for sinus tachycardia. For hypotension, she required temporary inotropic support. She was extubated 4 h after admission to the hospital, and the postextubation arterial blood gas values were normal.

Ten hours later, she developed precordial chest pain and shortness of breath. The pain was localized and reproducible in nature. Results of physical examination disclosed a pulse of 110 beats per minute, BP of 100/70 mm Hg, and a respiratory rate of 30 breaths per minute. She had bibasilar crackles and an S3 gallop. An ECG revealed T-wave inversions in leads 1, aVL, V3-V6. A transthoracic two-dimensional echocardiogram revealed diffuse hypokinesia of the left ventricle with an approximate ejection fraction of 25%. Left ventricular size was normal, and there was no evidence of pericardial effusion. A chest roentgenogram showed pulmonary edema. Serial tests for creatine kinase-MB isoenzyme were negative.

The patient was treated with bed rest, analgesic agents, and diuretics, and her symptoms improved. Subsequent ECGs showed diffuse and deep T-wave inversions and QT interval prolongation. These changes were more pronounced on day 3 of hospitalization, after which they started receding gradually (Fig 1). A repeat

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changes 1. ECGs ventricular abnormalities Day an and scan and months after been has has been developed prolonged myocardial depression following a brief episode of severe hypoxemia. The global nature of ventricular hypokinesis, the absence of cardiac enzymes, and the complete restoration of electrical and mechanical function of the heart strongly support the diagnosis of myocardial stunning in this patient. The chest pain was most likely musculoskeletal in origin even though pericarditis could not be excluded with certainty. Coronary spasm due to stress-related catecholamine surge is unlikely because of its rare occurrence and the absence of ST segment elevations.

A careful search of the English-language medical literature did not reveal any previous reports of myocardial stunning following respiratory arrest. This case illustrates that myocardial stunning may occur as a sequela following successful resuscitation from respiratory arrest. This condition can occur in young subjects without any preexisting coronary artery disease, and because it is reversible, it does not necessarily indicate a grim prognosis. Hence, we recommend that such patients should be closely observed for any signs of cardiac decompensation.

**References**


**Biopsy Evidence of Atrial Myocarditis in an Athlete Developing Transient Sinoatrial Disease***

Andrea Frustaci, MD, FCCP; Sergio Cameli, MD; and Paolo Zeppilli, MD

Atrial myocarditis causing transient sinoatrial disease (incessant atrial tachycardia alternating with sinoatrial pauses of up to 6 s in duration) in an athlete is reported. Diagnosis was undertaken by endomyocardial biopsy; biventricular and right atrial specimens were ob-

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