inotropic effects of catecholamines. Stressing this last topic, we would like to report what we experienced in our laboratory. After a protocol for coronary artery bypass without heart-lung machine on dogs, sudden reperfusion after 40 min occlusion of the left anterior descending artery resulted in ventricular fibrillation in three dogs. We started all resuscitation maneuvers adopted in Hospital Universitário Antônio Pedro, Niterói, Brazil, for heart surgery, including internal defibrillation and massage, adreneline, lidocaine, and bicarbonate. Before we stop them, 100 mg aminophyllin—an adenosine receptor antagonist—was administered as a last resource via the left ventricle apex. Internal defibrillation was tried again, and all three hearts presented sinus rhythm.

The events succeeding ischemia-reperfusion are varied; however, we agree that we witnessed a practical example of adrenergic blockade mediated by adenosine, and reversed by aminophyllin, in part by adenosine receptor antagonism, and in part by its phosphodiesterase inhibition effects. The use of aminophyllin has already been described in the same course of cardiopulmonary resuscitation by bradiarrhythmias.3

We conclude that adenosine blockade could be useful in cardiac arrest, but it must be further studied.

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REFERENCES

To the Editor:

Drs. Provenzano and Paschoal ask a valid question, "Is there a place for aminophylline in cardiopulmonary resuscitation?" The use of aminophylline in this situation, particularly in the dose ranges quoted, would be for the nonselective blockade of cardiac adenosine receptors. Many investigators believe that in a cardiac arrest, an accumulation of endogenously produced adenosine occurs as a consequence of cellular hypoxia. Adenosine has direct effects—smooth muscle vasodilatation, depression of sinoatrial and atrioventricular nodal automaticity, conduction—and indirect effects—antiadrenergic. These adenosine-mediated actions, obviously in the extreme, may cause or maintain circulatory collapse.

Wesley and Belardinelli showed in a porcine model that adenosine was a significant mediator of postdefibrillation conduction disturbances and hypotension. These actions could be attenuated effectively with the addition of an adenosine antagonist. In a second experiment, dipyridamole, an adenosine uptake blocker that potentiates the action of endogenously produced adenosine was added before inducing cardiac arrest. Marked electrophysiological depression and hemodynamic instability were noted in the postdefibrillation period as compared with control. This postdefibrillation instability was reversed with an adenosine receptor antagonist. Lerman and Engelstein reported that adenosine, through its indirect antiadrenergic effects, can raise the defibrillation threshold during ventricular fibrillation. This effect also could be successfully abolished with the addition of an adenosine receptor blocker.

Therefore, the reasons aminophylline is effective in restoring postdefibrillation electrical and hemodynamic stability in this report and others are because (1) it abolishes the adenosine-induced rise in the defibrillation threshold, (2) it reverses the negative chronotropic and dromotropic effects of adenosine, and (3) it attenuates adenosine-mediated vascular smooth muscle dilatation. Is there a place for aminophylline in cardiopulmonary resuscitation? I think there is, particularly in cases refractory to usual and standard care. The final answer will come with the completion of several controlled trials currently underway in the United States.

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Ethambutol-Induced Neutropenia and Eosinophilia

To the Editor:

Although hematologic adverse reactions due to antituberculous therapy have been reported now and then, the combination of neutropenia and eosinophilia is unusual. We would like to report such a case that results from ethambutol administration. This 87-year-old male patient was admitted to the hospital in March 1993 for management of reactivation of pulmonary tuberculosis. His other concomitant medical diseases also included chronic obstructive lung disease and β-thalassemia minor. He was initially administered a combination therapy of rifampicin, isoniazid, and ethambutol. This was followed by dramatic reduction of neutrophil count associated with progressive eosinophilia. Antituberculosis therapy was suspended in early May 1993 followed by recovery of the hematologic derangement. Bone marrow biopsy at this juncture revealed conspicuous infiltration of eosinophils, gross paucity of neutrophils and its precursors, and normal erythroid and megakaryocytic series. No evidence of leukemia or other malignant infiltration was seen. He was subsequently readmitted forloxacin and ethambutol in late May 1993 with the assumption that the blood dyscrasias were probably related to rifampicin or isoniazid.

However, this was proven not to be the case as showed by recurrence of neutropenia and eosinophilia. Ethambutol was then omitted from the antituberculosis regimen that subsequently was composed of ofloxacin, isoniazid, and rifampicin. The blood picture was normalized gradually and was stable with the afore