Pulmonary Hemorrhage in a Cocaine Abuser

To the Editor:

We read with interest the case report by Murray et al ("Diffuse Alveolar Hemorrhage Temporally Related to Cocaine Smoking", *Chest* 1988; 93:427-29) relating pulmonary hemorrhage and cocaine inhalation, and report on our experience with a similar case.

A 30-year-old white male was admitted to the intensive care unit because of rapidly progressive hypoxemia and diffuse pulmonary infiltrates on chest roentgenogram. He was a known abuser of intravenous narcotics and admitted regular use of cocaine by both the nasal and intravenous routes. Unlike the previously published case, our patient denied "free-basing". On admission, hematocrit was 18 percent with no evidence of external blood loss nor hemolysis.

Opportunistic lung infection was suspected and transbronchial lung biopsy attempted, but tissue was inadequate for analysis and the procedure was limited by the patient's increasing respiratory distress. An iron stain of the cell block of bronchoalveolar lavage fluid showed many hemosiderin-filled macrophages consistent with pulmonary hemorrhage. Special stains for *Pneumocystis carinii* and other pathogens were negative. The patient's subsequent course was one of rapid spontaneous resolution of hypoxemia and pulmonary infiltrates, and his hematocrit remained stable after transfusion. ANA was negative, and there was no hematuria or other evidence of vasculitis.

In light of the case report by Murray et al, it seems possible that our patient's process might also have been related to his cocaine abuse. He has regularly used cocaine in the past, however, as well as subsequent to hospitalization; if in fact this habit was etiologically related to his episode of pulmonary hemorrhage, some other factor (perhaps an adulterant in the drug) might be responsible. With the increasing use of "recreational" drugs in our society, a history of cocaine use should be sought in patients with unexplained pulmonary hemorrhage.

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ST Depression and Elevation in Vasospastic Angina

To the Editor:

We have read with great interest the recent article by Rechavia et al1 describing a patient with repetitive ventricular fibrillation preceded by alternating ST segment depression and elevation. The authors suggested that these ECG changes, confined to the precordial leads only, reflected subendocardial and transmural ischemia of anterior wall due to coronary spasm. In agreement with these interesting observations, our previous experience2 suggested that, in patients with variant angina, precordial ST segment elevation and depression occurring in the same patient may indeed reflect partial and total transmural ischemia due to coronary spasm and that sometimes these ECG changes may be associated with ST segment alternans during both ST segment elevation and depression. Although precordial ST segment depression seen in ambulatory monitoring does not necessarily reflect anterior wall subendocardial ischemia, it is unlikely in our case that these findings were due to "concealed" transmural ischemia affecting the opposed inferior wall, as suggested by Rozanski. Indeed, in our case the patient showed previous ST segment depression and elevation on precordial anterior ECG leads at rest during anginal attacks, indicating that the ECG changes seen on subsequent ECG ambulatory monitoring were due to anterior wall subendocardial ischemia. Therefore, our previous experience appear to be similar to that observed by Rechavia et al. The ECG changes in our case, however, frequently occurred but were of brief duration (1 min) and sometimes were characterized by alternans of both elevated and depressed ST segment. A recent clinical study has indicated that alternans of the elevated ST segment in patients with variant angina represents an index of the severity of ischemia and a precursor of lethal ventricular tachyarrhythmias.3 To the best of our knowledge, alternans of the depressed ST segment has never previously been documented in human subjects. Like alternans of the elevated ST segment, we believe that alternans of the depressed ST segment may represent a marker of the severity of ischemia (subtotal) associated with ventricular instability. In our case, the alternans phenomenon occurred with both elevated and depressed ST segment suggesting that ventricular instability was associated with severe myocardial ischemia (total and subtotal, respectively). The

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communications to the editor

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