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A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]



Hyperventilation and Myocardial Infarction*

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Chest pain that is associated with hyperventilation is often considered to be benign and noncardiac in nature. While not commonly recognized, hyperventilation can provoke coronary vasospasm. We report a man who presented with hyperventilation and developed myocardial infarction. In the setting of hyperventilation, chest pain and ST segment elevation, coronary vasospasm must be considered.

Up to 10 percent of patients seen by primary care physicians have symptoms attributable to hyperventilation.¹ Chest pain is a common but poorly understood complaint in these individuals.^{2,3} It has been thought to arise as a result of intercostal muscle spasm or strain or by diaphragmatic irritation from a distended stomach.⁴ Hyperventilation can produce T wave and ST segment changes on the ECG,^{5,6} but coronary angiography in these patients usually reveals normal anatomy.⁷⁻⁹

In addition to causing peripheral and cerebral vasoconstriction, hyperventilation has also been shown to cause diminished coronary blood flow.^{10,11} Oxygen delivery to the myocardium and other tissues is further decreased in alkalosis because of increased hemoglobin oxygen affinity according to the Bohr effect. In patients with known Prinzmetal's angina, hyperventilation has been observed to induce coronary vasospasm and myocardial ischemia.¹²⁻¹⁴

The following case report implicates hyperventilation in the pathogenesis of myocardial infarction in a man with near-normal coronary arteries and no prior history of angina.

CASE REPORT

A 59-year-old building contractor was brought to the Milwaukee County Medical Complex on October 18, 1985 with mild chest pressure, dyspnea, paresthesias of extremities and difficulty concentrating, of one hour duration. He had a history of intermittent atrial fibrillation and was taking digoxin and quinidine. In 1982, he had abnormal findings on exercise stress test, but a completely normal coronary angiogram. At presentation he was anxious, tachypneic, nauseated and diaphoretic. The initial ECG revealed normal sinus rhythm and a slightly prolonged Q-T interval of 0.38 s without ST segment changes. Electrolytes and chest x-ray film findings were normal. An initial arterial blood gas determination showed pH, 7.65; Pco₂, 13 mm Hg; Po₂, 125 mm Hg on room air. He was given 5 mg of diazepam by mouth and instructed to breathe into a paper bag. Approximately 50 minutes later he complained of increased precordial chest pressure. Repeat ECG (Fig 1) showed marked S-T segment elevation in leads V₃-V₆, and he was admitted to the coronary care unit.

He initially received little relief from sublingual nitroglycerin or nifedipine and was then treated with IV nitroglycerin and proprano-

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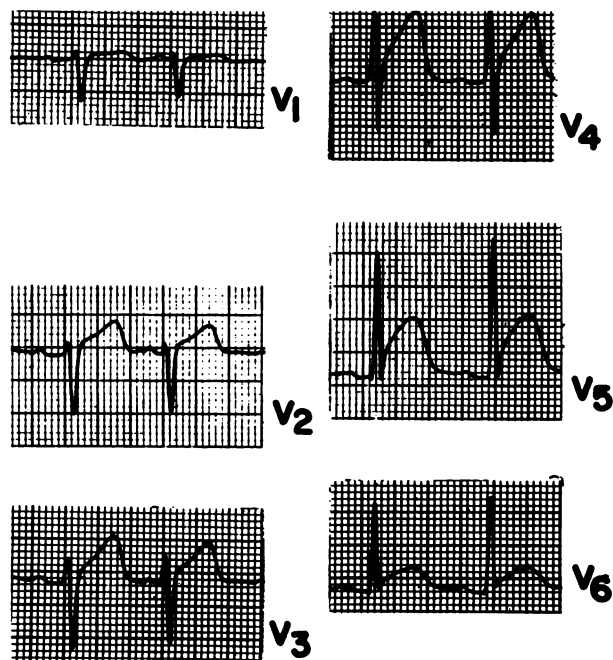


FIGURE 1. ECG obtained during hyperventilation and chest pain.

lol with resolution of his chest pain. At 12 hours, his CPK level peaked at 1,824 IU with 12 percent MB bands. He had a four-beat run of ventricular tachycardia on the night of admission, which was treated with lidocaine. An ECG the following day showed Q waves in leads V₁-V₄ (Fig 2). Cardiac catheterization on the fifth hospital day revealed normal hemodynamics. Other than a small 10-20 percent defect in the proximal left anterior descending artery, his coronary arteries appeared normal (Fig 3). There was a focal area of hypocontractility of the distal anterolateral wall near the apex. He was discharged on therapy with nifedipine, warfarin, aspirin, dipyridamole, digoxin and quinidine. One month later, a gated heart scan revealed a normal resting ejection fraction of 53 percent. He has remained pain free in the ensuing 18 months, but has developed recurrent atrial fibrillation.

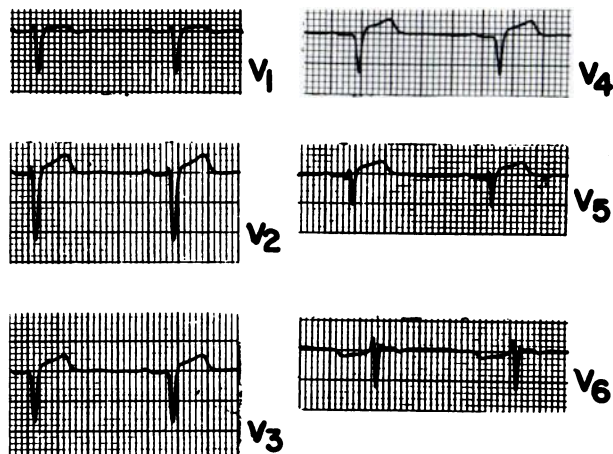


FIGURE 2. ECG obtained the second day of hospitalization.

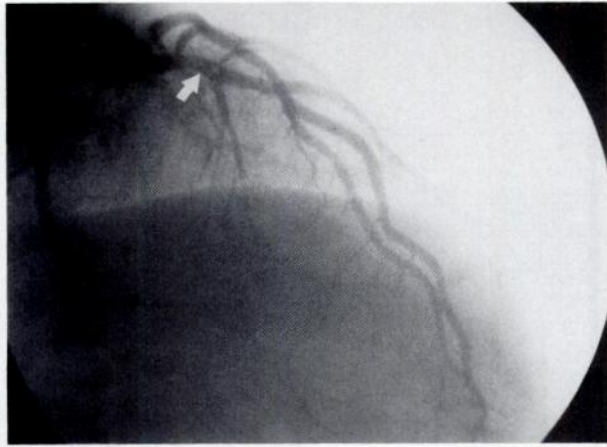


FIGURE 3. Coronary angiogram on the fifth day of admission. No significant stenosis is seen. The arrow depicts a small filling defect that may represent a thrombus.

DISCUSSION

This case describes a man, presenting with hyperventilation, who had a transmural myocardial infarction, but near normal coronary arteries five days later. Normal findings on coronary angiograms have been reported after myocardial infarction in 3 to 6 percent of those studied.^{15,16} In these cases, coronary vasospasm, thrombosis or platelet aggregates are thought to cause temporary coronary occlusion.

Yasue et al¹² first demonstrated that hyperventilation, in combination with Tris buffer infusion, caused coronary vasospasm. Preadministered diltiazem prevented vasospasm in four patients studied. Hyperventilation is more apt to provoke coronary ischemia in individuals with Prinzmetal's angina than those with classic exertional angina.¹³ Therapy of Prinzmetal's angina with vasodilator drugs can be assessed by serial trials of hyperventilation. There was good correlation between clinical remission of angina on therapy and lack of ischemic response to hyperventilation in one group of patients with Prinzmetal angina.¹⁴

The mechanism by which hyperventilation causes vasospasm is unclear. It has been proposed that during alkalosis fewer hydrogen ions enter the cell. This permits more calcium to enter the cell causing vasoconstriction.¹⁷ The autonomic nervous system may play a role as propantheline bromide, an antimuscarinic agent, has been shown to prevent ECG changes with hyperventilation.¹⁸ Circadian fluctuations in catecholamine levels might explain the observation that some patients have ischemic responses to hyperventilation only in the early morning.¹⁹

The effect of hyperventilation on blood coagulation is not well known. Platelets, *in vitro*, exhibit increased aggregability to epinephrine and collagen at a physiologically alkaline pH.²⁰

In the case reported herein it is not possible to ascertain whether myocardial infarction occurred because of vasospasm, thrombosis or platelet aggregates. There is substantial evidence that hyperventilation can provoke coronary vasospasm, but cause and affect cannot be established here. Individuals with Prinzmetal's angina appear to be more susceptible to hyperventilation-induced myocardial ischemia. Calcium channel blockers and efforts to terminate hyperventilation such as sedation, reassurance, rebreathing and

instruction in relaxation techniques should be utilized. Further studies are needed to define the scope of hyperventilation-induced myocardial ischemia in the general population.

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