Asystole Following Treadmill Exercise in a Man Without Organic Heart Disease

Jerome L. Fleg, MD, Abraham V. K. Asante, MD

- A 52-year-old man without evidence of organic heart disease by clinical and extensive noninvasive examination experienced an 11-s episode of asystole ten minutes after completing a maximal treadmill test. Four years previously, symptomatic sinus bradycardia and hypotension had also followed cessation of treadmill exercise. This case illustrates that vagally mediated complications of treadmill exercise occurring in persons without apparent heart disease may be potentially life-threatening and can be elicited on repeated testing. (Arch Intern Med 1983;143:1821-1822)

Major complications from exercise testing occur in less than 0.1% of tests and generally consist of myocardial infarction or ventricular fibrillation. Such complications are confined largely to persons with overt or suspected coronary artery disease. Asystole occurring in association with exercise testing is not mentioned in either of two large American surveys with a combined total of almost 700,000 exercise tests.1,2 A Swedish study of 50,000 tests, performed mostly using bicycle ergometers, identified two cases of asystole, one in a patient with aortic stenosis and the other in a patient with uremia and chronic bronchitis.2 We describe herein a 52-year-old man without organic heart disease who experienced an 11-s episode of asystole ten minutes following cessation of maximal treadmill exercise.

REPORT OF A CASE

A 52-year-old man in excellent health, except for a five-year history of mild hypertension controlled by 100 mg/day of chlorthalidone, was admitted to the Gerontology Research Center, Baltimore, on July 21, 1982, for his biennial examination as a participant in the Baltimore Longitudinal Study on Aging. On a prior visit four years earlier, he had a transient episode of sinus bradycardia of 35 to 40 beats per minute accompanied by mild hypotension one minute following cessation of maximal treadmill exercise, during which he achieved a maximal heart rate of 195 beats per minute without ischemic ECG changes. These abnormalities resolved spontaneously within five minutes. Two days later he underwent submaximal treadmill exercise ECG and thallium scintigraphy, achieving a heart rate of 160 beats per minute without complications; both ECG and thallium test results were normal.

At the time of his current admission to the Gerontology Research Center, he had no cardiovascular symptoms. Physical examination was normal except for a standing BP of 138/100 mm Hg in the left arm. Resting ECG showed sinus rhythm at 70 beats per minute with a PR interval of 0.22 s but was otherwise unremarkable. The patient underwent treadmill exercise with a modified Balke protocol for 11 minutes at a constant speed of 3.5 mph (approximately 8.6 metabolic equivalents). He achieved a heart rate of 186 beats per minute and a BP of 198/106 mm Hg at maximal effort. No symptoms, ectopic beats, or repolarization abnormalities were noted during exercise. By six minutes following exercise cessation, the heart rate was 106 beats per minute and 0.7 to 1.6 mm, J-point depression had developed in leads 2, aV3, and V6, each with a slowly upsloping ST segment. Three minutes later, marked sinus arrhythmia was noted and the PR interval increased to 0.29 s in those beats terminating shorter RR intervals. Ten minutes after cessation of exercise, the heart rate suddenly decreased from 70 to 30 to 35 beats per minute, and, 15 s later, asystole occurred (Figure). Asystole lasted 11 s and was accompanied by loss of consciousness. A precordial thump was unsuccessful but spontaneous electrical activity returned 2 s postthump, followed by another 3.8-s pause before sinus bradycardia at a rate of 30 to 35 beats per minute was noted. Sinus rhythm at 90 beats per minute resumed about 15 s later and persisted throughout the subsequent two-hour monitoring period. No symptoms or ECG signs of ischemia developed during the subsequent 24 hours. Results of a complete blood cell count and standard blood chemistries obtained the following morning were normal except for a serum potassium level of 3.2 mEq/L. An M-mode echocardiogram demonstrated normal, left-ventricular cavity size, wall thickness, and systolic function. Twenty-four-hour ambulatory ECG begun at this time was unremarkable, as was the response to carotid sinus massage. At the completion of his two-day examination, the patient was referred back to the care of his private physician and was instructed to avoid strenuous exercise. Three months after his asystolic episode, the patient remains free of cardiovascular complaints.

COMMENT

Although postexercise vagal reactions have been said to occur in 0.2% of exercise tests,4 we have found only one detailed report of asystole resulting from such a reaction, occurring in a healthy 35-year-old physician approximately one minute following cessation of bicycle exercise.4 Our case illustrates several important points. Postexercise vagal

Continuous V6 ECG rhythm strip recorded ten minutes following cessation of treadmill exercise. Paper speed is 10 mm/s. Sudden slowing of heart rate from 70 beats per minute to 33 beats per minute begins with nonconducted P wave (first arrow). Asystole ensues, interrupted after nine s by chest thump (second arrow) and terminated after 11 s by what appears to be junctional escape beat. Another junctional beat follows 3.8 s later before sinus rhythm returns. ECG contact was lost for several seconds at this point, as patient was being moved. When ECG monitoring resumed, patient was in sinus rhythm at rate of 90 beats per minute.
reactions can be re-elicited in a predesised person, in this case, after an interval of four years. Such reactions are not limited to the early recovery period from exercise. Had our patient been released from the laboratory after five or six minutes of postexercise monitoring, the norm in many laboratories, his collapse might erroneously have been attributed to ventricular tachycardia or fibrillation. Although our patient made an uneventful recovery, a patient with coronary artery disease might experience severe ischemia or infarction from the profound hypotension occurring during such an episode. Finally, submaximal exercise testing did not evoke a vagal reaction in our patient only two days following his initial reaction four years previously, suggesting that activation of this exaggerated vagal reflex depends in part on the intensity of the exercise effort. Persons with documented symptomatic postexercise vagal reactions should be advised to avoid maximal exercise and should probably be instructed to self-administer atropine at the onset of such a reaction occurring outside the medical setting.

References

Polycystic Kidney Disease and Polycythemia Vera
Occurrence in a Patient Receiving Hemodialysis

Nancy B. Jermanovich, MD

- Symptomatic erythrocytosis developed in a 59-year-old man with polycystic kidney disease (PKD) while he was receiving maintenance hemodialysis. Major clinical and laboratory data suggested a diagnosis of polycythemia vera (PV), despite a normal serum alkaline phosphatase level and leucocyte count. Secondary erythrocytosis, related to chronic hypoxemia and increased erythropoietin production, was excluded by appropriate laboratory studies. Despite previous documentation of secondary erythrocytosis in patients receiving hemodialysis, to my knowledge, PV has not been described in this population.

(Ann Intern Med 1963;143:1822-1823)

Although a relative elevation in the hematocrit secondary to increased erythropoietin (Ep) production has been documented in patients with azotemia and polycystic kidney disease (PKD), to my knowledge, primary erythrocytosis has not been described in patients requiring maintenance hemodialysis. Clinical and laboratory findings compatible with a diagnosis of polycythemia vera (PV), four months after coronary artery bypass surgery, developed in a dialysis patient with PKD.

REPORT OF A CASE

A 59-year-old man with end-stage renal disease secondary to PKD had been maintained by routine hemodialysis without complications since 1975. He had required no transfusions and his hematocrit reading remained in the mid-30s without androgen therapy. His medical history included coronary artery disease, since 1978, and hypertension. He had had no history of PV before renal failure. The patient had no evidence of lung disease, alcoholism, tobacco use, or sleep disturbances. The patient's family history was positive for PKD in four of his five siblings.

In 1979, the patient was hospitalized for evaluation of unstable angina. A cardiac catheterization disclosed severe atherosclerosis involving two major coronary vessels. Coronary artery bypass surgery was performed without incident in April 1979. During the ensuing months, the patient began to complain of subjective headaches, increasing pruritus, and dizziness. Medications at that time consisted of propranolol hydrochloride, calciotriol, digoxin, folic acid, ferrous sulfate, multivitamins, and aluminum hydroxide.

Physical examination showed an alert, normotensive man in no distress, with grade 2 hypertensive retinopathy, a systolic ejection murmur, bilaterally enlarged kidneys, and a palpable spleen 2 cm below the right costal margin. His lower extremities were edematous and cyanotic, with evidence of peripheral vascular insufficiency. Representative laboratory test results showed the following values: serum urea nitrogen, 67 mg/dL; serum creatinine, 12.2 mg/dL; calcium, 4.8 mg/dL; phosphorus, 5.6 mg/dL; and serum alkaline phosphatase, 9 IU/L (normal, 4 to 13 IU/L). Liver function studies were normal and hepatitis B surface antigen was negative. The C-terminal parathyroid hormone level was elevated 11,992 pgEq/mL (normal, 150 to 375 pgEq/mL), but the intact parathyroid hormone was normal, 284 pgEq/mL. Roentgenographic bone survey demonstrated minimal evidence of secondary hyperparathyroidism. Hematologic profile disclosed a hemoglobin level of 16.7 g/dL, hematocrit reading of 53.8%, and a reticulocyte count of 2.0% (normal, 0.1 to 1.4%), with an absolute reticulocyte count 109 x 10^10/L (normal, 17 to 77 x 10^10/L). The WBC count was 8.4 x 10^9/L, without evidence of basophilia, and the platelet count was 250 x 10^10/L. Serum viscosity was normal at 1.4. Total blood volume was elevated, with an RBC volume of 38.3 mL/kg (normal, 25 to 30 mL/kg) and a plasma volume of 33.2 mL/kg (normal, 39 to 49 mL/kg). Leukocyte alkaline phosphate score was normal at 105 (control, 158). The serum Ep level by hemagglutination-inhibition assay was reduced to 10 mU/mL (normal patients, 7 to 36 mU/mL; patients receiving maintenance dialysis with hematocrit readings of 18% to 34%, 25 to 50 mU/mL). Arterial blood gas study results on room air were as follows: pH, 7.30; PCO2, 33 mm Hg; PO2, 110 mm Hg; and O2 saturation, 96%, without evidence of hypoxia. Liver-spleen scan demonstrated splenomegaly without cystic enlargement, despite some cystic areas in the liver.

A phlebotomy was performed twice with subjective improvement in symptoms (Figure). The hematocrit reading, however, remained in the mid-40s until the patient's unexpected death five months later.

COMMENT

Secondary PV associated with increased serum Ep production has been demonstrated in several different renal diseases, ie, PKD, renal cell carcinoma, and hydronephrosis. Many patients with end-stage renal disease secondary to PKD maintain a relative erythrocytosis in the absence of blood transfusions or androgen therapy, despite impaired renal function. Similar increases in erythrocytosis mass occur in maintenance dialysis patients with either hepatitis or acquired cystic disease associated with chronic glomerulonephritis. Elevated levels of serum Ep without spleno-