Images in cardiovascular medicine
Pacemaker failure caused by hyperkalemia

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A 70-year-old female patient was admitted to our hospital because of recurrent episodes of fainting and syncope. She had a history of mitral valve disease and congestive heart failure and had undergone surgical mitral valve replacement with a mechanical prosthesis 6 years earlier. Because of a slow heart rate in the presence of atrial fibrillation, she had also undergone a VVI pacemaker (PM) implantation 2 years earlier. Her drug therapy included enalapril (20 mg daily), spironolactone (100 mg daily), furosemide (25 mg daily), digoxin (0.25 mg daily), and warfarin. The 12-lead ECG at admission (Fig. 1) showed the absence of any atrial activity, and a slow regular rhythm (47 b/min) with wide QRS complexes and a right bundle branch block pattern, suggesting an escape idioventricular rhythm. Regular spikes (rate 70/min) of the VVI PM were also visible, indicating both sensing and capture failure. Routine laboratory tests revealed increased creatinine (2.4 mg/dl) and potassium (7.0 mEq/l) serum levels. Intravenous calcium gluconate (1 g) and furosemide (20 mg), intravenous glucose-insulin infusion, and Kayexalate enema were administered. Normalization of potassium serum levels (to 4.8 mEq/l) was observed after 2 hours. The ECG (Fig. 2) showed now spontaneous irregular rhythm (atrial fibrillation) with narrow QRS complexes and frequent short periods of a normally functioning VVI PM rhythm.

This case illustrates the occurrence of an impairment of both sensing and capture functions of a ventricular PM as a consequence of acute hyperkalemia, which was caused by the occurrence of a mild renal failure in a patient treated with both an angiotensin-converting enzyme inhibitor (enalapril) and a potassium-sparing diuretic (spironolactone). Hyper-

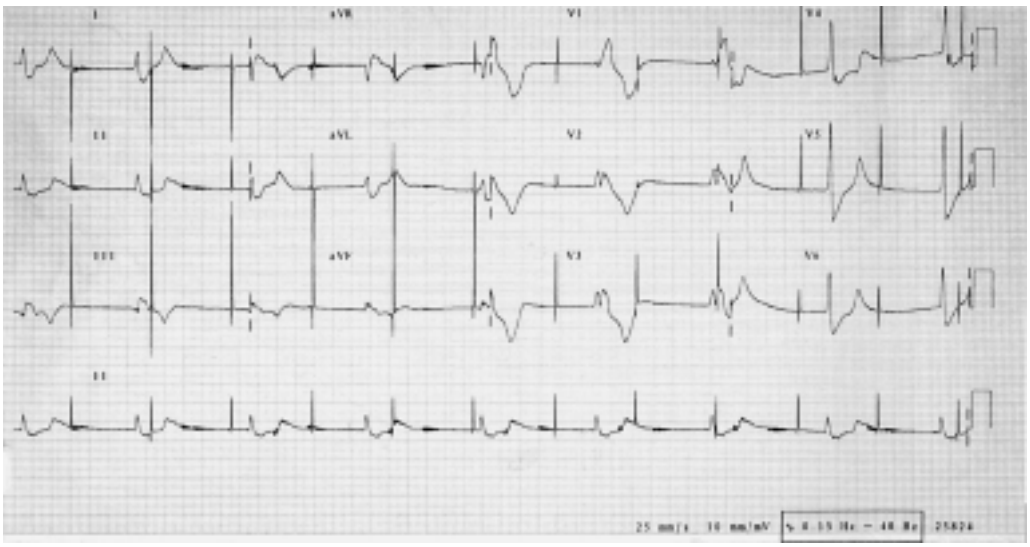

Figure 1.

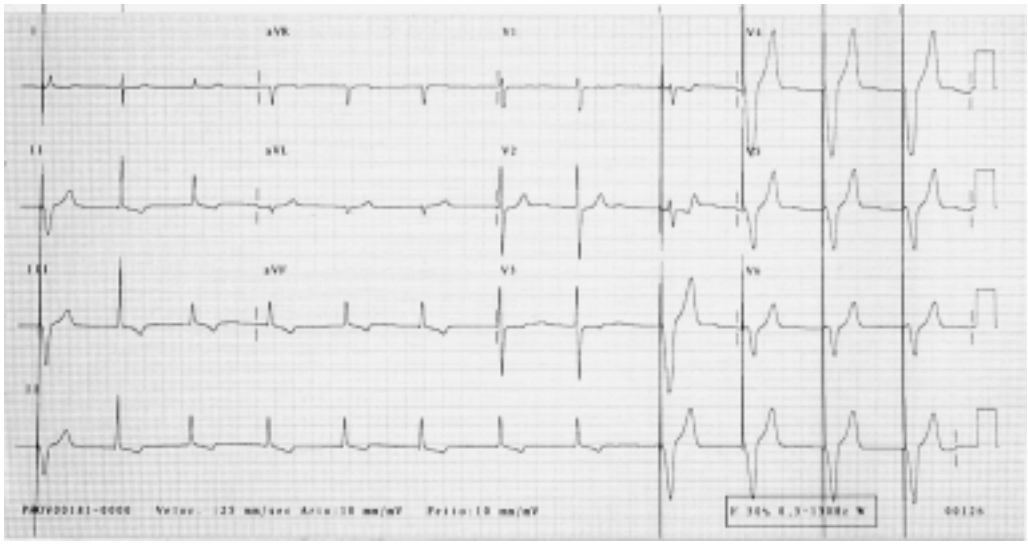

Figure 2.

kalemia is known to increase the threshold of myocardial excitability, and therefore can prevent ventricular activation by the electrical PM impulses[1]. In accordance, several reports showed PM capture failure caused by hyperkalemia[2-4]. Of note, at variance with previous reports, also a PM sensing failure was observed in this case, suggesting that action potentials generated by myocardial cells were of such a low amplitude to be under the PM sensing threshold, thus preventing their perception by the sensing electrode of the PM lead.

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