Aldosterone Blockade in Patients With Acute Myocardial Infarction

To the Editor:

In Dr Bertram Pitt’s excellent editorial, “Aldosterone blockade in patients with acute myocardial infarction,”1 the first word in the next-to-last line of the left column on page 2525 should, I believe, read “hyperkalemia” rather than “hypokalemia.” In addition, in discussing the possible mechanisms by which aldosterone blockade might result in a decrease in sudden cardiac death, one might have included the rather old demonstration that administration of intravenous potassium canrenoate, an antialdosterone agent, to patients with acute myocardial infarction reduces ventricular extrasystoles and ventricular fibrillation,2 as well as the more recent observation that aldosterone alters myocardial repolarization by increasing monophasic action potential duration.3

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Response

Thank you for pointing out the error in regard to hyperkalemia rather than hypokalemia. The reference by Denis et al1 is relevant, and the effect of aldosterone blockade in preventing ventricular arrhythmias and sudden cardiac death may be related to its effects in improving myocardial norepinephrine uptake,2 as well as several other factors mentioned in the editorial.

The reference by Tillmann et al3 is, however, problematical as the effect of aldosterone on monophasic action potentials is a nongenomic (rapid) effect which is not blocked by spironolactone. It is likely, therefore, that the beneficial effects of both spironolactone and eplerenone on sudden cardiac death are due to their genomic effects, possibly by the mechanisms suggested in the editorial.

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